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BRAIN HERNIA: A POSTOPERATIVE COMPLICATION IN OTOLOGY*†

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LOS ANGELES

Brain hernia is a rare complication in mastoid surgery, and it has received scant mention in American literature. It seemed proper, therefore, to review the outstanding work of the foreign and American writers, to combine their ideas and experiences with those of the author in a recent case, and present the following compilation of facts concerning this rare condition.

DEFINITION AND TERMINOLOGY

The term "brain hernia" is employed here to denote a herniation of meningeal and brain substance through a defect in the dura mater and into the mastoid wound.

The following terms have been used synonymously in the literature: cerebral hernia, brain fungus, brain prolapse, and encephalocele—brain hernia, however, is given preference here because:

(1) "Cerebral hernia" occasionally leads to confusion as to whether the protrusion is cerebral or cerebellar;

(2) "Brain fungus," a descriptive term used because of the fungoid appearance of the semistrangulated brain substance and granulations, is sometimes erroneously considered to be an etiologic term;

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(3) "Brain prolapse" denotes a "falling down or in" rather than a protrusion as described above;

(4) "Encephalocele," a protrusion of brain through a cleft in the skull, is best limited, in otolaryngology, to those rare congenital conditions so completely reported in 1936 by William E. Adams.[1]

HISTORICAL DATA

H. Caboche[4, 5] in 1901 prepared a thesis on brain hernia in otology and in 1902 published the first comprehensive, illustrated study of the condition. A. Ya. Galebski[9] in 1911 contributed another complete survey. The treatises of these two authors remain to this date the most exhaustive and comprehensive in otologic literature. Caboche reported two cases, neither of which had brain abscess. Of Galebski's two cases, one had brain abscess.

Hugh Jones[13] in 1902 reported the anatomical findings in a case of brain hernia associated with temporosphenoidal abscess.

Schifone[28] in 1904 reported the results of his brain surgery, and his article has been quoted frequently since.

Koch-Bergmann's[16] case in 1906 followed trepanation for a brain abscess which was not present.

Krause[21] made a report in 1907 concerning meningeal adhesions and has been widely quoted.

Fr. Reinking[24] in 1909 made a thorough review and collected 122 cases of hernia in which abscess was present in 68. This report included nasal cases. He reported five cases of his own.

W. S. Bryant[3] in 1909 demonstrated a case to the New York Academy of Medicine, Section of Otology. His report includes a short discussion by Lederman and Kopetzky.

L. W. Dean[6] in 1910 advocated marked changes in the handling of brain abscess cases with a resultant decrease in the occurrence of hernia and also a decrease in mortality rate.

M. Leriche[18] in February, 1913, reported a case in which abscess was not present.

Lieutenant Colonel Percy Sargent,[26] 1915, in his "Remarks on Fungus Cerebri," referred to traumatic cases. However, his article contains a clear discussion of the pathogenesis of brain hernia.

Dutheillet de Lamothe[7] in 1923 reported a case of hernia following surgical intervention for lateral sinus thrombophlebitis. The report is accompanied by a photograph of his case.

M. Sargnon[27] in 1924 discussed the treatment of brain hernia and reported two cases—one following trepanation for abscess where none was found, the other occurring during the postoperative course of a lateral sinus thrombophlebitis. M. Leriche,[19] who in 1916 advocated extensive resection of the bony collars of these hernias, discussed the paper.

J. E. J. King,[15] 1924, in his "Treatment of Brain Abscess by Unroofing and Temporary Herniation of the Abscess Cavity," gave valuable information concerning the management of brain hernias in general.

Luigi Torchiana[30] in 1924 produced brain hernias experimentally in dogs. His article contains photographs of the gross pathology. He also gave a complete review of the literature but failed to mention the most outstanding early paper, that of Caboche.

Henry Wieder[31] in 1928 recorded one case of hernia complicating lateral sinus thrombosis. Wieder refers to several authors and their various methods of controlling herniations.

F. Fremel[8] in 1929 reported one case with abscess and gave a histopathological description of the herniated mass.

Martin Hirsch[12] of Breslau in 1930 reported one case with a brain abscess and thrombophlebitis and another case with thrombophlebitis alone. He quotes Neuman, who had sixteen cases of brain hernia complicating brain abscess.

Carlos Goris[10] in 1931 recorded a case following surgical intervention for a lateral sinus thrombophlebitis and reported the findings of three biopsies.

Aurelio Slaviero[29] in 1932 produced experimental brain hernias and reported their histopathology.

Guillermin, Kliszowski and Favre[11] in 1936 reported a case following a trepanation in which a brain abscess was found.

M. Jean Razimbaud[23] in 1936 reported one case following a lateral sinus operation, and he discussed the treatment and possible means of prevention.

K. Neumark[20] in 1935 reported a hernia which occurred after a mastoid operation at the age of one. This case had no brain abscess. He relates his observation of this case over a period of ten years and describes his experience at surgery on the same side ten years after the formation of the hernia.

P. Roques and P. Huard,[25] February, 1937, in an illustrated article gave the complete gross pathology of a traumatic brain hernia. They quoted Beriel[2] whose pathological work was done in 1916.

PATHOGENESIS

General Considerations

As pointed out by Caboche[5] and Galebski,[9] postoperative hernias in otologic surgery may be divided into two groups:

1. Immediate hernias—those which occur immediately upon incising the dura.

2. Secondary hernias—those which occur from two to several days after the dura has been opened.

The mastoid operation, of course, produces the opening in the skull; however, two other conditions must obtain in order for a herniation of brain to take place. These are:

1. An opening in the dura.

2. Increased intracranial pressure.

It is essential that these conditions co-exist as the presence of one alone is insufficient to produce a hernia of brain substance such as defined above.

The opening in the dura results either from (1) trepanation for brain abscess, or (2) accidental injury, usually during surgical treatment of a diseased lateral sinus.

The increase in intracranial pressure, as shown experimentally by Slaviero[29] and also as demonstrated conclusively in clinical cases, does not result from an increase in spinal fluid formation, but does result from an increase in the brain volume. This increase in brain volume is either (1) a mechanical increase, resulting from the volume of the abscess or tumor, or (2) an edematous increase resulting from infection or irritation.

The size of the dural opening deserves some attention. A small dural defect does not preclude the formation of a large hernia. This has been proved in many reported cases, and in the case of the author the opening in the dura did not exceed five millimeters in diameter, while the ultimate diameter of the hernia was approximately four centimeters. It is important to remember that a small dural opening acts as a constricting collar which causes strangulation of the herniating tissue. Razimbaud's[23] two cases demonstrated this point rather effectively. His first case had a very large dural opening which re-

sulted from an operation for a depressed skull fracture; the resultant hernia appeared one week postoperatively, and when seen five years later, it had receded to below the level of the bony opening. In his second case, a very small opening was made during a lateral sinus investigation. In this case a large strangulated hernia completely filled the retro-auricular groove, and five years later, although it was completely covered with skin, it had not reduced any in size. King's[15] work also is conclusive upon this point. He deliberately made a large defect in the dura, completely unroofed the abscess and allowed the remaining portion of the abscessed cavity to herniate through the cranial defect. He stated that with proper treatment the hernia in such a case recedes and becomes completely covered with epithelium within two and a half to three months from the time of the operation.

Immediate Hernias

Immediate herniation upon incising the dura is evidence of a mechanical increase in the brain volume and this usually presumes the presence of abscess or tumor. Caboche[5] stated that the immediate hernia is usually small and disappears after evacuation of the abscess. He stated also that a subtentorial abscess will produce a much greater rise in the intracranial pressure than one of equal volume in the supratentorial region. It is evident, therefore, that immediate hernias more commonly follow cerebellar trepanations. Caboche[5] explained, however, that while an immediate herniation in this region is evidence of a subtentorial lesion, the same deduction cannot be made concerning supratentorial herniations. A large cerebellar lesion may cause an internal hydrocephalus or "ventricular hydrops" sufficient to produce a herniation of the temporal lobe if a trephine were made in this region and would, therefore, *not* be an indication of a true cerebral abscess. This point is proved by a case of Reinking[21] in which there was a bilateral temporal lobe herniation in the presence of a cerebellar abscess and in the absence of cerebral pathology other than an internal hydrocephalus. The reverse is not true because the tentorium is so tough and unyielding that an abscess in the supratentorial region will cause no appreciable increase in the tension of the posterior fossa.

Secondary Hernias

Secondary hernias have a somewhat different pathogenesis. Sargent's[26] description of the formation of traumatic hernias may well be adapted to secondary postoperative herniations. He wrote:

"The escape of brain matter from the cranial cavity, which is frequently seen in the earliest stages, is due to the sudden increase of pressure caused by the blow and to traumatic cerebral edema which immediately follows. If no additional factors were to come into play to sustain and increase this pathological intracranial tension, any initial protrusion would disappear with the subsidence of the traumatic edema and no fungus cerebri would form. Infection in the wound, however, results in the onset of secondary inflammatory edema which keeps up and increases the general intracranial pressure. This leads to a further protrusion of the softened brain through the dural opening which becomes more and more tightly plugged, and consequently the drainage of the damaged and septic brain is increasingly interfered with. This interference causes a still further rise in intracranial tension so that a vicious circle is established."

Secondary postoperative hernias may be subdivided clinically into three groups:

1. Cases in which an abscess is present.

2. Cases in which trepanation for abscess was negative.

3. Cases associated with lateral sinus disease. For the practical consideration of pathogenesis we may combine groups 2 and 3.

Caboche[5] stated that secondary hernias, occurring in the presence of brain abscess, are due to a combination of mechanical pressure and the extensive softening of the tissue around the abscess.

In the cases following accidental opening of the dura or trepanation where no abscess is found, it is the author's belief that the trauma produced, which may be compared to "the blow" described above by Sargent,[26] is probably not sufficient as a rule to cause any appreciable increase in intracranial tension. However, either one or a combination of two other factors may produce such a hernia. These factors are:

1. Infection extending in from the already infected mastoid cavity.

2. Edema resulting from the pressure of dressings and from other irritating trauma.

Whereas Slaviero[29] experimentally produced edematous herniations with no evidence of infection, nevertheless it is reasonable to suppose clinically where we have operated through an infected area, that infection is always present, though it may be well defined and of low grade. The predominance of the infection factor was clearly shown in the author's case. Sufficient pressure was exerted by the

lateral sinus packs to cause cerebellar symptoms; these packs were removed after seven days, and normal appearing brain cortex could be seen through the dural defect; yet herniation did not occur until seventeen days after the pack removal. Had pressure been the main factor, herniation should have occurred almost at once after the pack removal.

So we see that Caboche's[5] statement, "Il y a donc là dualité pathogenique; infection et action mécaniques," is adaptable to all secondary postoperative hernias, the "action mécaniques" being the abscess in the one case and the brain edema in the other.

Recapitulation

In regard to the dural opening:

1. Dural opening alone is insufficient to cause a hernia.

2. The size of the opening is important because of the strangulation caused by small openings.

In regard to the increase in intracranial pressure:

1. In immediate hernias the cause is mechanical.

2. In secondary hernias there is a pathogenic duality—infection and mechanical action.

PATHOLOGY

Experimental

Torchiana[30] and Slaviero,[29] using dogs, produced secondary hernias by the injection of sterile olive oil—one-half to three cubic centimeters—into the lateral ventricle, but their work does not clearly state the size of the dural opening; however, it is quite clear that they did not use pressure bandage and it is also clear that they operated through a clean field. Torchiana published photographs of the gross pathology at various stages, and his pictures clearly demonstrate the strangulation of the hernia at its neck, and he stated, "* * * these herniæ are really fungi and are inactive." Slaviero's[29] work deals with the histopathology of hernias ranging from 36 hours to 103 days in age. He found that both the choroid plexus and the ependymal layer were normal in all sections, thereby ruling out the formation of excessive spinal fluid in the ventricular system. He found no evidence of infection in any case. The 36-hour section showed normal cortex and white substance of the brain with considerable edema and perivascular round cell infiltration. From this period on through the 103rd day section, the nervous tissue and the

vascular tissue were seen to go through all stages of degeneration. The protoplasm became very scarce, there were necrotic cells with deeply staining nuclei, and there appeared to be much intercellular fluid. By the 103rd day the section showed what Slaviero[29] described as "merely a ghost or shadow of all cell structures." Microphotographs of the early stage and the 47-day stage accompany the article, and show the cortex, white substance, neuroglia and dilated blood vessels.

Clinical

The pathology here can be most satisfactorily covered by answering the questions which Caboche[5] asked of himself:

1. Of what tissue is the hernia composed?

From the reports of Galebski,[9] of Beriel,[2] of Fremel,[8] of Goris,[10] and of Roques and Huard,[25] we may conclude that the microscopic pathology is the same clinically as that in the experimental cases described above. In other words, the hernia is composed originally of brain tissue.

2. What is the fate of the meninges?

Caboche[5] answers this question clearly. The dura may adhere to the bony edge of the wound and form a collar for the hernia, or it may extend a few millimeters upon the hernia and gradually lose itself. The two layers of the arachnoid are so thin and delicate that they are certainly destroyed by the rub of the dressings. As to the pia mater, it directly accompanies the hernia and in large hernias may form a sac, very fragile and very dangerous, since it contains vessels which may cause fatal hemorrhage. Roques and Huard[25] stress the importance of the meningeal adhesions about the opening of the wound, which process prevents or tends to prevent a wide spread meningitis. One of their gross sections shows a point where there was a lack of adhesions of the dura and a subdural abscess resulted. The early work of Krause[21] also emphasized the importance of dural adhesions.

3. What is the content of the hernia?

It may be—

a. exclusively brain tissue—clean or infected;

b. pus from the main abscess;

c. prolongation of the lateral ventricle in temporal lobe hernias.

This was true in the traumatic case of Roques and Huard.[25]

4. What is the anatomical evolution of the hernia?
Histological studies have now proved that in most cases which are strangulated, the tissue becomes degenerated, atrophic, cicatricial, and eventually covered with a thin layer of normal skin. In the non-strangulated cases, a portion, or all, of the tissue recedes and re-enters the cranial cavity and is epithelialized.

CLINICAL COURSE

Caboche[5] stated that the immediate hernia usually disappeared after the evacuation of the abscess. Therefore the discussion to follow concerns the secondary hernia only. Within a few days, occasionally as long as several weeks postoperatively, there appears in the mastoid wound a soft granular mass which one at first mistakes for exuberant granulations. This soft mass increases in volume very rapidly, fills the wound, and usually protrudes from one and a half to two or more centimeters beyond the bony margin. Goris[10] described his strangulated hernia as a brawny mass of granulations which seemed to be more firm than ordinary granulations. Dutheillet de Lamothe[7] said: "* * * this mass is elastic and irreducible. It makes a hernia through an orifice which is much smaller than itself, the rigid collar of which strangulates the exteriorized portion which mushrooms out. The tissue is reddish without presenting any trace of necrosis."

The only immediate subjective symptoms are those attributable to the mechanism producing the hernia, none resulting directly from the hernia itself.

At this early stage, if the proper attention is given to cleanliness and prevention of trauma, the following termination may be expected. The markedly strangulated hernias do not reduce perceptibly in size, while those which are less strangulated, may recede to the level of the bony wound or even further. In either case the hernia becomes covered with a thin layer of normal skin. This process is completed within a period ranging from six weeks to eight months; usually, however, it is accomplished within two to three months.

Caboche[5] lists the following terminations and possible complications:

1. Healing. The surface becomes covered with healthy granulations and the neighboring skin gradually and completely covers it. After the hernia has become covered with skin, one may find directly under this thin epidermis layer, cerebrospinal fluid; sometimes enough to distend the cicatrix and form a veritable cerebrospinal lake.

2. Sphacelation or sloughing. This is rare. Galebski[9] stated that this may cause meningitis by penetrating the ventricle or by sloughing into the subarachnoid space at the edge of the wound.

3. Hemorrhage. This is rare. It results from a rupture of a large pial vessel.

4. Adhesions between the reduced hernia and the scalp covering it.

The following complications, mentioned by Galebski[9] and also by Roques and Huard,[25] should be added to the above:

5. Abscess formation in the hernia.

6. Diffuse encephalitis.

7. Inclusion of the ventricle in the hernia.

The escape of cerebrospinal fluid in these cases deserves some discussion. The following is a quotation from Keen's[14] Surgery: "A protrusion of the brain covered by intact soft parts carries no risk of life and may even save it by warding off fatal compression; a protrusion which becomes exposed, which granulates, necroses, and leaks cerebrospinal fluid, is almost always fatal." This latter may be true of badly infected traumatic cases, but it is not necessarily true of the cases secondary to otologic surgery. Bryant,[3] Wieder,[31] Goris,[10] and most recently Guillermin, Kliszowski and Favre,[11] all report the escape of cerebrospinal fluid in their cases without a fatal result.

The question arises as to the various neurological symptoms which have occurred in these cases months and even years later. From the known pathology, as described earlier in this paper, we can make only one deduction: There are no subjective symptoms attributable to the hernia itself; any neurological symptoms present must be a result of a pathological disturbance of the intracranial anatomy and physiology.

It is significant that the fatal terminations and serious complications have usually occurred in those cases where an abscess was located and where excessive manipulation and trauma had occurred; this, regardless of the site of the hernia.

K. Neumark[20] in 1935 reported an interesting clinical experience. He had performed a mastoid operation and a trepanation of the dura on a patient during the first year of life. A brain hernia resulted. This healed and became covered with skin. During the

ten years postoperatively the patient was observed closely and found to be normal mentally and physically. At the age of 10 he developed an otitis media and an intense headache. At surgery, through the old mastoid wound, the hernia was found to prevent the drainage of pus from the antrum region. Trauma during a postoperative treatment caused temporary intracranial signs, but these promptly disappeared and the patient again made an uneventful recovery.

There is another hernia which differs slightly from the definition used in this paper, but which is of sufficient rarity and interest to warrant mention. This is the dural covered hernia.

Bryant[3] in 1909 reported a case, in which a radical mastoidectomy had been performed previously, and upon his examination of the ear, he found what appeared to be a polyp. However, this polyp turned out to be a dural-covered herniation with a discharging sinus leading into a temporal lobe abscess.

Leriche[18] in 1913 had a most unusual case. A 15-year-old boy had a mastoid operation in which the dura of the middle fossa was uncovered. He made an uneventful recovery and was well for two years. He presented himself again two years later with an otitis media, intense headache, increased intracranial pressure, positive Kernig, and pulsation over the old operative scar. When the old mastoid wound was reopened, Leriche found a cerebral hernia. The dura was incised and the pressure relieved. The patient's symptoms promptly receded. Leriche felt that the recurrence of the otitis had caused an increase in intracranial pressure which in turn brought about the formation of a cerebral hernia through the old bony defect resulting from the first operation. Whatever the explanation, it might be well for us all to keep this case in mind when we reopen old mastoidectomy wounds.

FREQUENCY AND PROGNOSIS

In reviewing the literature, it is difficult to determine how many of the brain hernias resulting from abscess and abscess trepanation were protrusions into the mastoid wound and how many resulted from trepanations in some site other than the mastoid wound. This point, however, is not of great clinical importance. The important clinical point is that this complication of abscess surgery, which was common many years ago, has become very rare during the past 25 years. This unquestionably is due to the more rational surgical methods which will be mentioned later. The mortality rate of these cases was extremely high, the deaths being due to the causes enumer-

ated in the preceding section, mainly meningitis. The cases occurring within recent years report a more favorable prognosis as regards the hernia itself, if properly treated; fatalities occurring only as a result of the primary disease.

Carl W. Rand[22] reports that in his neurosurgical experience, secondary herniation following trepanation for abscess (whether or not abscess is found) through a clean field is seldom, if ever, encountered.

Secondary brain hernia following injury to or disease of the lateral sinus is very rare—there having been only seven cases reported. These have been reported by Lederman,[17] Dutheillet de Lamothe,[7] Wieder,[31] Hirsch,[12] Goris,[10] Razimbaud,[23] and the present author.

It is interesting that there were no fatalities in this entire group. All of them followed the clinical course outlined above, in spite of the fact that some of them were repeatedly subjected to resection, and they repeatedly recurred. Razimbaud[23] in 1936 made the following note regarding the prognosis of hernia of the brain: "Brain hernia, either as a suppurative or non-suppurative complication, perhaps does not carry the dark prognosis formerly believed." This, no doubt, is a result of the more rational, the more aseptic, and the less meddlesome methods of handling.

PREVENTION OF HERNIA

L. W. Dean[6] in 1910 revolted against the textbook and time-honored methods of dealing with abscesses because of the high mortality and high incidence of infected hernias. He reported his experience with these hernias and found them to be due to extensive dural opening and excessive manipulation of the abscess cavity; the damage done to the limiting wall of the abscess cavity and the making of a large opening combined two mechanisms of hernia formation. He then laid down the following dictum which practically eliminated the occurrence of hernias in these cases: He advised that in an abscess with a discharging sinus (one in which there is a perforation in the dura through which the abscess is discharging), the protective adhesions about the dural opening should not be disturbed, and that the wall of the sinus in the brain, being a good protective one, should be treated with extreme care. He felt that unless this discharging sinus was present, the exploratory trepanation should be made through a clean field.

Carl W. Rand[22] states that in exploring for a possible brain abscess, if one trephines through a clean field (not through the mastoid wound) and no abscess is found, the trephine has been made from a clean field into a clean field, thus eliminating the possibility of carrying in infection. No abscess being found, the wound is closed and no hernia results. If abscess is found, the draining outward onto a clean field is of no serious consequence. Exploring through the mastoid wound for possible abscess, means trephining through an infected field into a possible clean field. The older reports show that where this was done, hernias were rather common as a result of infection being carried into the brain substance.

The almost complete lack of hernias following gentle aseptic trepanation through a clean field and the high frequency of hernias accompanying the old method of extensive manipulation through an infected field, suggest rather conclusively the advisability of operating through a clean field as advocated by Rand[22] and Dean,[6] except in the unusual case where a discharging sinus is present.

The logic of avoiding trauma and infection is also based upon the work of Krause[21] who demonstrated the protection of dural adhesions and of Schifone[28] who found that if no infection was present, no secondary hernia would form.

Hernias during the course of lateral sinus disease should be avoided by careful technique during curettement, excision, or other surgical interference in this region. The possibility of necrosis of the visceral dura of the lateral sinus must also be constantly kept in mind.

TREATMENT OF HERNIA

The early treatment of a brain hernia should be directed toward (1) the avoidance of trauma, (2) the prevention of infection, and (3) the lowering of intracranial pressure. Very light, non-irritating dressings should be used. Galebski[9] stated that, clinically, the lack of parallelism between the quantity of spinal fluid after lumbar puncture and the size of the hernia, speaks for the fact that the increase in spinal fluid is only one link in the chain of causes of increased intracranial pressure. Therefore, in secondary hernias, too much must not be expected from lumbar punctures, and they should be done with extreme caution. The intravenous administration of hypertonic solutions, such as 50 per cent glucose, perhaps is a more rational procedure for lowering a pressure which is caused mainly by brain edema, and it is certainly much safer than lumbar puncture.

Among therapeutic agents which have been used—caustic solutions, alcohol, x-ray, galvanocautery—none have been effective.

Compression acts only to increase the causative mechanism, as one cannot expect to push the hernia back through the strangulating dural collar.

Removal is to be condemned because of the possibility of causing a meningitis, of spreadiing the encephalitis, of causing hemorrhage, or of possibly opening the lateral ventricle. Strangely enough it has been employed in a few cases with no ill effects. However, the herniation recurred and usually was larger than before.

Caboche[5] in 1902 concluded that in the great majority of cases the hernia cures itself, and all that matters in the treatment is antiseptic precaution.

The hernia becomes covered with skin without any grafting being necessary.

The advisability of later operations, such as resection of the bony collar suggested by Leriche[12] in 1916, or the various plastic procedures, depends entirely upon the conditions existing in each individual case.

REPORT OF A CASE

Case 1.—E. H., white, female, six years of age. Admitted to the Contagious Ward, November 13, 1936, with a diagnosis of septic scarlet fever.

November 13, 1936:

History: Sore throat and headache beginning on November 9, 1936. Onset of vomiting November 12, 1936. Generalized rash November 13, 1936. No ear history.

Examination: Temperature 104.4 degrees. Pulse rate 120. Respirations 26. Generalized maculopapular rash. Large red tonsils with a rather dirty membrane covering them. The left ear drum was gray and dull, and the right ear was reported to be filled with wax.

Throat Culture: Hemolytic streptococcus. Negative for diphtheria.

Blood Wassermann and Kahn: Negative.

Blood Count: White blood count, 14,000. Polymorphonuclears, 85 per cent.

Red blood count, 5,300,000. Hemoglobin, 88 per cent.

November 16: 75 cc. immune serum given.

November 17: 50 cc. immune serum given.

November 18: Purulent discharge was noted for the first time from the right ear; temperature 106 degrees. Blood culture—negative.

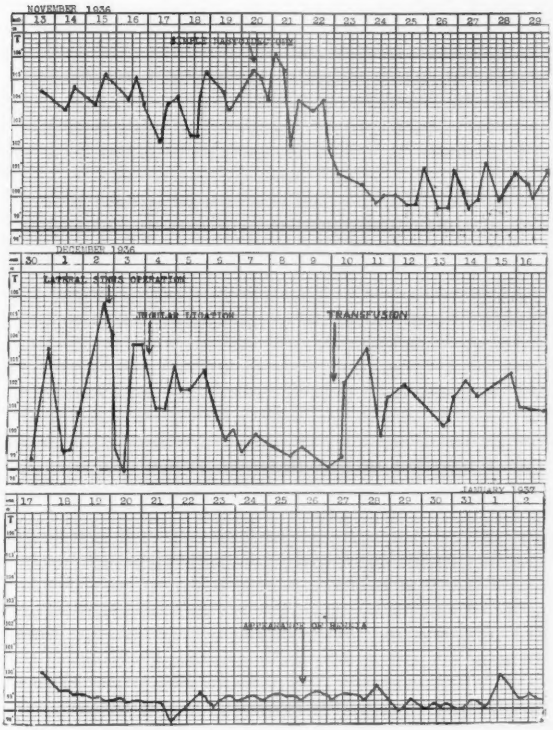

Fig. 1. Temperature chart.

Ear, Nose and Throat Consultation: "Scarlet fever of over a week's duration. Right otitis media—duration ? ? Temperature ranging from 102 to 106 degrees during the past six days."

Examination—Left ear: Canal clear. Drum dull grap. All landmarks recognized.

Right ear: Canal shows redness and swelling of the skin; there is grayish yellow material at the bottom of the canal and no definite landmarks can be recognized. There is periosteal thickening over the right mastoid process.

Throat: Large red palatine tonsils.

Nose: Purulent discharge seen posteriorly and superiorly.

X-ray: Suggestive of cell destruction with periantral clouding on the right. Left mastoid is well pneumatized and clear.

Conclusion: There is an infectious process going on in the mastoid, and if the sepsis continues, would advise mastoidectomy.

November 20:

The temperature having continued to fluctuate between 103 and 105.4 degrees, a simple mastoidectomy was done.

Considerable periosteal thickening. Thick bony cortex. No break in the cortex and no bleeding through the cortex. There was marked periantral necrosis with a small amount of pus. The sinus plate was quite necrotic and beneath it there was pus under pressure. The sinus dura was thickened and covered with necrotic granulations. The definitely pathological sinus wall was not disturbed except for wide exposure, and the establishment of drainage of the entire sinus region.

Diagnosis: (1) Mastoiditis. (2) Perisinus abscess. (3) Lateral sinus thrombophlebitis.

Culture from the mastoid: Hemolytic streptococcus.

Postoperative course was quite satisfactory until December 1, 1936. She received 400 cc. of citrated blood on November 22, and another blood culture was reported as negative.

December 2:

Spinal fluid: Negative culture; colloidal benzoin 000001110000000; globulin negative; quantitative chloride, 740 milligrams per 100 cc.; quantitative sugar, 66 milligrams per 100 cc.

White blood count, 19,750. Polymorphonuclears, 86 per cent.

December 2: Lateral sinus operation.

The mastoid was reopened, the lateral sinus was traced backward to normal appearing dura, the sinus was opened and free bleeding obtained from both ends. A section of the lateral sinus wall was removed in an effort to prevent later canalization; this resection was somewhat difficult because of an exceedingly bloody field; the sinus was then collapsed, and the bleeding controlled. This was only accomplished after exerting great pressure with the iodoform packs.

December 3:

White blood count, 16,800. Polymorphonuclears, 90 per cent.

December 4:

Ligation of the right internal jugular vein just above the facial vein. This was done because of continued sepsis.

December 9:

Temperature reached normal on this date, after a progressive decrease since the ligation. The lateral sinus packs were removed and no bleeding occurred. At

Fig. 2. Appearance of the brain hernia December 29, 1936, a few days after its initial appearance.

the site of what was previously the lateral sinus, could be seen a small opening about five millimeters in diameter, and through this opening one could plainly make out the pale pinkish appearance of the brain cortex. It was evident, therefore, that during the removal of a section of the lateral sinus, a small portion of the visceral layer was accidently removed, leaving a complete dural defect of the size mentioned above.

Following the surgery and packing of the lateral sinus, there was persistent projectile vomiting; marked nystagmus upon looking to the left; drowsiness, listlessness and moderate signs of meningismus. These began to disappear immediately upon removal of the packs.

December 19:

Following a transfusion of 200 cc. of blood on December 10, the patient ran a febrile course until December 17. Spinal fluid culture was negative, and blood cultures were negative. However, by December 19, the patient had markedly improved; there was complete disappearance of all intracranial symptoms, and the white count had decreased to 9,400.

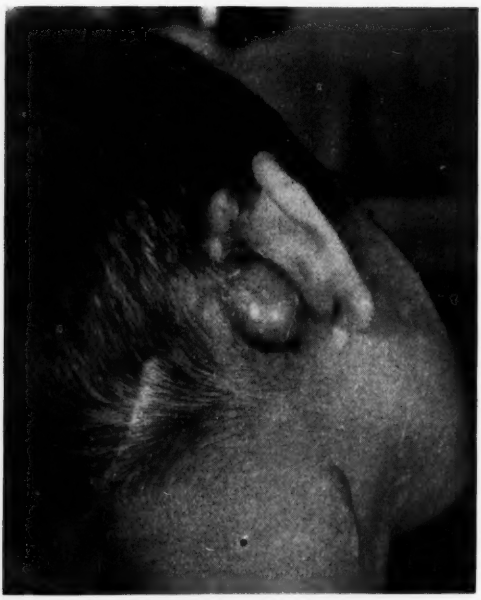

Fig. 3. Appearance of the brain hernia February 26, 1937, after it had been completely epithelized.

December 26:

Twenty-four days following the lateral sinus operation, and seventeen days following removal of the lateral sinus packs, there first appeared in the wound what seemed to be excessive granulations.

December 27:

By this date, the mastoid wound was filled with a mass which protruded about three centimeters above the bony level, as shown in Fig. 2. It was round, soft, reddish, looked like a mass of exuberant granulations, and did not pulsate.

December 29:

Patient examined by Dr. Carl Rand, who made a diagnosis of "brain fungus" and described it as a mass of new tissue, completely filling the mastoid wound.

Diagnosis: Brain hernia.

Treatment and Clinical Course:

No treatment other than mildly antiseptic dressings were employed on the hernia. The patient's condition remained excellent; there were no intracranial

symptoms after December 19, 1936. A very low-grade middle ear infection persisted, from which staphylococcus aureus was repeatedly cultured. The edges of the mastoid wound were quite healthy, there was no discharge from the mastoid wound, and epithelization from the skin edges gradually took place.

February 26, 1937:

Epithelization complete. (See Fig. 3.) By this date, approximately two months after its appearance, the hernia was completely covered with a thin layer of skin.

August, 1937:

The appearance of the retroauricular mass was the same as in Fig. 3, taken in February, 1937.

Comment:

Accidental injury to the visceral wall of the lateral sinus caused the dural defect. The mechanism of strangulation by a small opening was clearly shown. The extreme pressure and the irritation of the lateral sinus packs, while sufficient to cause intracranial symptoms, did not produce the mechanism of the hernia. This statement is based upon the appearance of normal brain tissue at the time of the pack removal, and the fact that the herniation did not occur until seventeen days later. In other words, the hernia appeared only after the brain substance had been directly exposed to the infection of the mastoid wound for several days.

CONCLUSION

From the foregoing study, it may be concluded that brain hernia in mastoid surgery is, in the majority of cases, an avoidable complication, and the prognosis is dependent upon the co-existing pathology and the methods of treatment.

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XXVII

CARDIO-INHIBITORY AND VASOMOTOR REFLEXES FROM THE NOSE AND THROAT*

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It is well known that changes in heart rate, blood pressure and respiration can be brought about reflexly by stimuli applied to the mucosa of the respiratory tract. The early experiments were suggested in part by the inhibition of cardiac action and respiration resulting from the administration of inhalation anesthetics to experimental animals (Kratschmer,[11] Francois-Franck[4]). Reflex cardiac inhibition evoked by trigeminal stimulation in the human has been studied by Russetzki.[12]

In the series of experiments to be described, reflex cardiac inhibition and blood pressure changes will be considered in some detail and respiratory effects mentioned only briefly. The work began as a study of the effects of tobacco smoke on the nasal mucous membranes, but the intensity of the reflex reactions turned the direction of subsequent experiments toward a clarification of the reflex mechanisms. Old experiments have been repeated and extended for the purpose of analysis.

Methods: In preparation for the experiments a machine for controlled smoking of cigarettes, or controlled administration of other vapors, was designed and constructed. The suction device, whose function was to "take a draw," consisted of a metal bellows operated by a suitably shaped and timed cam driven by a constant speed electric motor. In the machine there were actually four of these puffing bellows connected to the driving mechanism in parallel, but in practice it was found convenient to use only one at a time. The driving mechanism was fitted with an adjusting device by which the size of the puff could be given any desired volume from 5 to 35 cc. (The average human puff from a cigarette is about 35 cc. taken in about two seconds of suction.) In addition to the puffing bellows, there was a large metal bellows operated by another cam. Its purpose was

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to dilute the vapor with a desired volume of air. The volume and concentrations of the vapors were adjustable.

Rabbits were used in all animal experiments. In those trials in which vapors (ether, acetic anhydride, ammonia, cigarette smoke) were employed as stimulating agents, the application of the substance was done in a manner that prevented its reaching the lungs, thus assuring a minimum degree of absorption. Animals were prepared in two different ways. In the first experiments they were tracheotomized, the opening in the trachea being in the form of a slit. Before an experiment a suitably curved glass cannula was passed into the upper end of the trachea, through the larynx and into the pharynx. It was necessary to extend the cannula this far in order to prevent interference to the passage of the vapor by closure of the glottis, or sometimes the soft palate. The inferior part of the trachea was open to the air for respiration. Some tracheotomized animals were kept as long as four or five weeks, but they were difficult to maintain in good health, especially if used for experiments.

Another method of preparing animals for the application of gas to the nasal mucous membranes without allowing it to reach the lungs was developed, and the rabbits so prepared could be maintained in good health indefinitely and used in experiments not requiring further surgery as often as desired. A window was made in the dorsum of the nose connecting one side of the nasal cavity with the outside air. The window was prevented from closing by a soft rubber plug. After two weeks or more the animal was ready for experimentation. The end of a rubber tube was fitted into this window during the experiment. The vapor was passed through the window into the nose and out via the nostril on that side. The posterior passage was blocked with vaselined cotton during the experiment to prevent the possibility of inhalation of the substance. In order to be sure that the sense of smell did not affect the reactions, the cranium was opened and the olfactory stalks sectioned. Other items of method will be briefly described in connection with the experiments in which they were used. Either general or local anesthesia was employed during all surgical procedures.

Results: Stimulation of the nasal and pharyngeal mucous membranes of the rabbit with certain vapors gives rise to powerful respiratory, cardiac and vascular reflex responses. The cardiac and respiratory reactions are easily observed in a dramatic qualitative manner by palpating the heart and observing respiratory movements when ether is placed in contact with the end of the nose. Heart action and respiratory movements stop before any of the vapor is

inhaled. The heart then begins to beat slowly, at about 20 to 40 per cent of the normal rate, and continues this slow rhythm as long as thirty or forty seconds, after which it will quickly accelerate to its normal pace. Respiratory movement remains at a standstill during the period of great slowing. The passage of any of the vapors used as stimuli (ether, acetic anhydride, ammonia, cigarette smoke) through the nose or nose and pharynx of the rabbit gives rise to responses similar to those described, if in sufficient concentration. The description is of a maximal response. The quantitative relation that exists between concentration of vapor and degree of response as shown by diminution in heart rate is given later. The passage of a volume of room air equal to that of the vapor mixtures used in the tests is without demonstrable reflex effect.

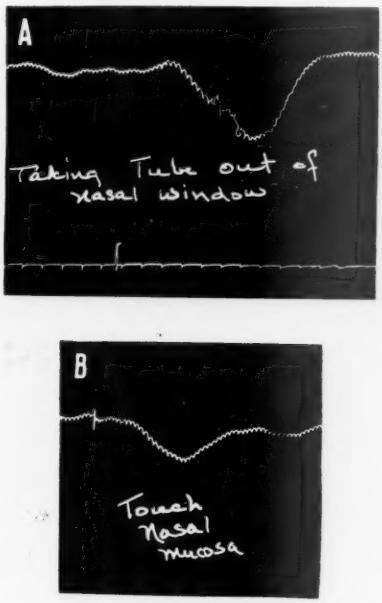

Fig. 1. Reflexes evoked by mechanical stimuli to the nasal mucosa.

A. Mercury manometer record of carotid mean blood pressure and pulse. Stimulus: removal of soft rubber plug from nasal window. B. Response to roiling probe on sensitive mucous membrane inside window.

Confirming Francois-Franck,[4] it was found that the cardio-inhibitory effects of all these various kinds of stimuli could be eliminated by (1) sectioning the vagi, (2) administering a dose of atropin sufficient to block vagal activity, or (3) inducing a sufficient degree of anesthesia. He showed that the cardiac inhibition is reflex and that the vagus nerve is the centripetal pathway.

In many experiments mercury manometer records of the carotid mean blood pressure were made. In some of them no general anesthesia was used, the cannulation being done after local subcutaneous infiltration with novocain. The reactions shown in Figs. 1 and 2 are from such an experiment. This animal had been prepared with a nasal window nine months previously and his responses were among the most sensitive seen in the whole series. Fig. 1-A shows

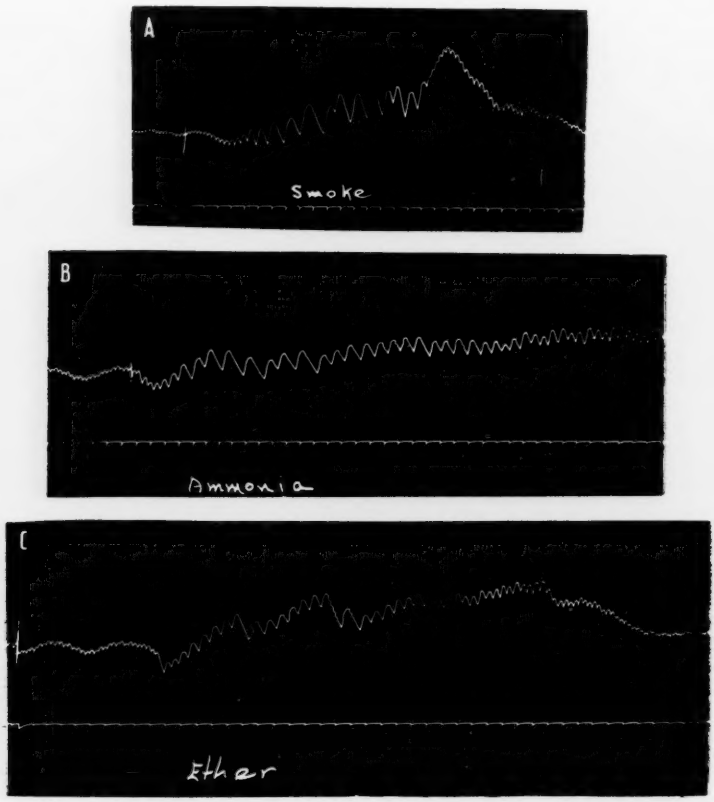

Fig. 2. Responses to vapor stimulation. A. 10 cc. of cigarette smoke diluted with 200 cc. of air; 100 cc. of mixture blown through nose in three seconds. B. A few drops of aqua ammonia held before nostrils five seconds, not touching. C. Cotton pad, wet with ether placed on end of nose about seven seconds. Sensitivity of animal had decreased appreciably when this test was made. Decrease was determined by repetition of smoke stimulus.

a fall in blood pressure of 38 mm., accompanied by a 27 per cent diminution of the cardiac rate. The stimulus was the removal of the plug from the window. Since the plug was soft rubber and was coated with vaseline, its removal should not be considered a severe stimulus. B was made by rolling a probe around the orifice of the window.

Fig. 2 from the same sensitive unanesthetized animal shows reactions to chemical stimuli. In each tracing it may be seen that

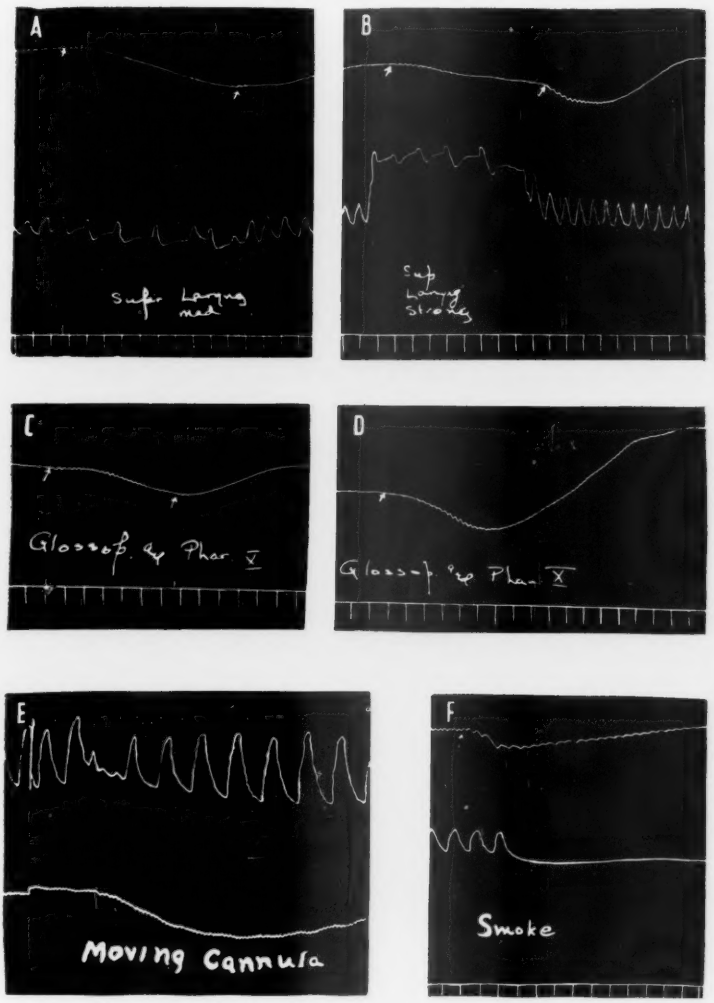

Fig. 3. A, B, C and D, Thyratron shocks, 60 per second. A. One and a half times threshold voltage applied to the superior laryngeal nerve. B. Thyratron shocks to superior laryngeal nerve four times threshold. C. Thyratron shocks to the glosso-pharyngeal nerve, and the pharyngeal (vagus) branch which joins it, one and a half time threshold. D. Thyratron shocks to the glossopharyngeal-pharyngeal, four times threshold. E. Turning cannula in larynx, touch stimulation. F. Another stimulation by smoke showing great diminution in heart rate with pressure below normal level throughout.

the first part of the response is a turning downward of the arterial pressure and slowing of the heart. While the pulse becomes very greatly slowed reaching a rate about 20 per cent of normal the arterial pressure, after the first dip, does not continue to fall but gradually rises a few mm. The cardiac inhibition (and activity of a vaso-depressor mechanism probably) did not last as long in the cigarette smoke experiment as in the other two. As the heart rate suddenly speeded toward normal a peak appeared in the blood pressure curve showing that a pressor mechanism was active. It had, evidently, been acting in antagonism to the depressor influences. When they were removed it was able to manifest its presence in this emphatic manner. The pressure sometimes remains below the normal or original level throughout the period of slowing as is shown in tracing F of Fig 3.

Rapidly repeated induction shocks applied to the sensory nerves supplying the mucosa of the respiratory tract evoked responses which were of some aid in the study of mechanisms. Stimulation of the infraorbital nerve will evoke cardiac slowing, but it will do so only if the shocks are strong enough to bring forth a considerable degree of pain reaction also. The same is true of the nasal branches of the maxillary nerve. The nerves to the pharynx and larynx appear to contain a relatively much smaller component of pain fibers to confuse the result.

The reflex results of stimuli just slightly above the reflex threshold applied to the superior laryngeal nerve are shown in Fig. 3-A, and the responses to similar weak shocks to the glossopharyngeal together with a pharyngeal nerve (from the vagus) with which it is joined are shown in Fig. 3-D. In each case there is cardiac slowing, and a lowering of blood pressure. There is no evidence of a pressor component. When these same nerves were excited by stronger shocks, however, evidences of pressor activity became manifest. The superior laryngeal record in Fig. 3-B is a bit complex. There is slowing of the heart and a very gradual fall in pressure which is continued during the period of stimulation. At the end of the stimulation period there is a sharp dip in blood pressure and new slowing of the heart followed by a quick return of the rate to normal and a rise of pressure to slightly above the original level. There were probably antagonistic mechanisms in action in this case, but they are more clearly shown in Fig. 3-D. Stimulation of the glossopharyngeal-pharyngeal pair with strong shocks evoked a marked diminution in heart rate and arterial pressure during stimulation. At the cessation of stimulation the pressure rose past

the original level to a point 44 mm. higher. This probably means that the nerve contains a considerable mixture of pressor and depressor fibers. When both were excited the depressor influence was the stronger, but on stopping stimulation the pressor activity outlasted the depressor and there was a "rebound" rise. The weaker shocks in A and C excited only the depressor and cardio-inhibitory fibers. Stronger stimuli excited these and pressor fibers also. Gently moving the cannula in the larynx elicits a lowering of pressure with little effect on rate (Fig. 3-E).

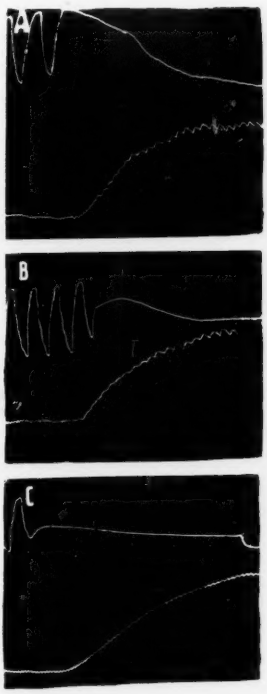

Fig. 4. Stimulation by acetic anhydride vapor (similar responses are seen with ether, smoke and ammonia).
In A and B cardiac slowing followed upon rise in arterial pressure. Light urethane anesthesia. In C the anesthesia was deeper and cardiac effect is much less.

The reactions to stimuli are not always depressor. In an animal whose depressor responses are not delicately sensitive, a stimulus of irritant vapor in the nose may evoke a large rise in pressure with or without appreciable inhibition of the heart. Rises of pressure upon stimulation of pharyngeal and nasal mucosa by acetic anhydride vapor are shown in Fig. 4. In A and B there is a large diminution in pulse rate accompanying the increase in pressure. In C the diminution in rate is much less, and in some other experiments no change of rate was seen. Urethane anesthesia was used in the experiments. The differences in cardiac response are due to different degrees of narcosis.

Analysis of the reactions points to independent neural mechanisms for each of the several factors exhibited: cardiac inhibition, vasomotor depression, vasomotor excitation, and cardiac inhibition which is secondary to increased pressure. Records from the most sensitive animals showing cardiac slowing accompanying or preceding a sharp diminution of pressure definitely eliminate the idea of a necessary carotid sinus reflex (Heymans, et al.[9]) or other cardio-inhibitory response which is dependent upon a primary rise in pressure.

The cardiac inhibition precedes by several cycles the ensuing fall in pressure in some cases. Also the various records fail to show a parallelism between degree of pressure drop and degree of cardiac inhibition. It seems to follow, therefore, that primary cardiac inhibition and vasomotor depression must be evoked by mechanisms that are independent of each other and which may be excited together in varying degrees or separately. The distinctness of mechanisms which bring about the different responses is to be expected in the light of other studies on sensory and reflex phenomena. Heinbecker, Bishop, and O'Leary[8] demonstrated that the skin senses of touch, pain, warmth, and cold were mediated by distinct groups of afferent fibers, and Harris[7] identified separate fiber groups for different skeletal muscle reflexes.

The primary cardio-inhibitory and vaso-depressor responses to small chemical or tactile stimuli in the nose are most delicate reactions, eliminated by quite light narcosis, and even when the arterial cannula is inserted with local anesthesia any great degree of surgical manipulation diminishes or eliminates it. Experiments requiring surgery must be done quickly with little struggling. Even so, some animals are less sensitive than others and stronger stimuli are required. In these less sensitive ones the depression is masked by a pressor response and the slowing of the heart is probably a combination of both primary and secondary cardio-inhibitory influences. Cardio-inhibitory and vaso-depressor responses to electrical excitation of the superior laryngeal and pharyngeal nerves are somewhat more durable in the rabbit than are the nasal responses, and Brodie and Russell[2] evoked similar reactions from the pulmonary branches of the vagus in anesthetized dogs. The reflexes of the dog may not be as vulnerable to anesthesia as are those of the rabbit. The pressor response survives surgical anesthesia in the rabbit but the carotid sinus and depressor nerve effects do not. However, they are more durable than the depressor and primary cardio-inhibitory effects. Since the reflexes which would be most important as factors in the production of syncope are eliminated by narcosis, reports in the literature must be examined with this fact in mind. Since Gording's rabbits were under full surgical anesthesia, his paper is entirely without value as regards reflex action.

Different degrees of cardio-inhibitory sensitivity found in totally unanesthetized healthy rabbits with no recent surgical trauma are shown by the graphs (Figs. 5 and 6) of the relation between concentration of stimulating vapor (cigarette smoke) and per cent slowing of the heart rate. These two rabbits were litter mates (ac-

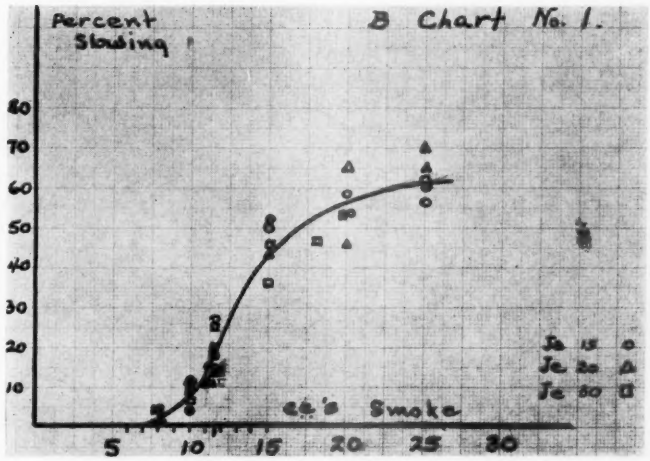

Fig. 5

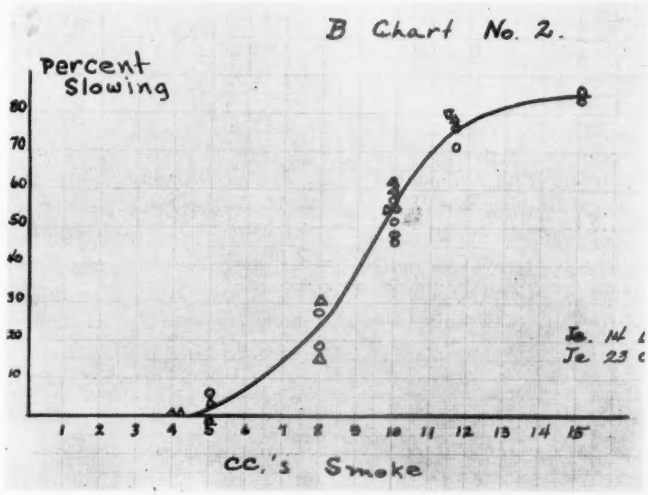

Fig. 6

Figs. 5 and 6. Curves showing quantitative relations between concentration of stimulating substance and degree of response in rabbits that were said to be litter mates. Note the difference in magnitude of abscissa scales. Note also repetition of response on different dates.

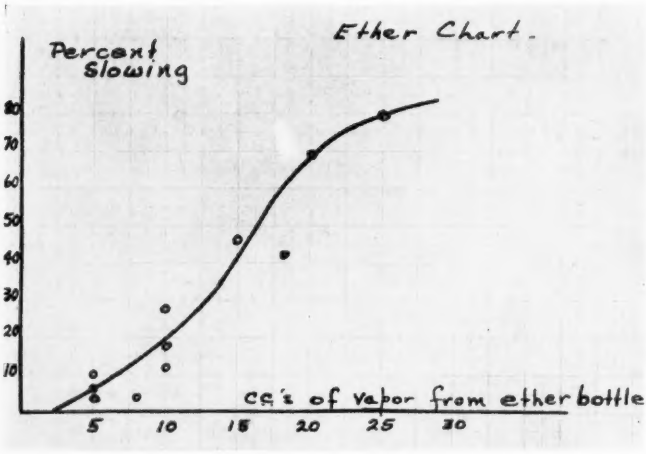

Fig. 7. Curve made with ether vapor stimuli showing that the response is not peculiar to one kind of stimuli.

cording to the breeder). Their nasal windows were made on the same day and six weeks elapsed before the first tests. All of these measurements were made from electrocardiograms. Though the sensitivities of the two animals differed greatly from each other, each was quite constant in his own responses as shown by tests that were weeks apart.

Respiration is suspended by all chemical stimuli strong enough to have much inhibitory effect on the heart. The respiratory movement stops in whatever phase may exist at the instant of stimulation (Fig. 4). Only when the stimulus is intense does the rabbit assume the position of expiratory spasm described by Kratschmer as characteristic of the response. Sneezing is inconstant even in unanesthetized rabbits. Only a small percentage of the animals will sneeze.

Human Responses: In the absence of direct evidence it would obviously be unjustifiable to assume that the reactions found in rabbits indicate those to be expected in the human under similar conditions. In some experiments with normal human subjects the nasal mucosa was subjected to irritation while electrocardiograms were being recorded (Fig. 8). The stimuli in some experiments were insufflations into the nose of ammonia vapor strong enough to be painful and evoke a flow of tears. In other experiments a very large draw of cigarette smoke (about 50 cc.) was taken and imme-

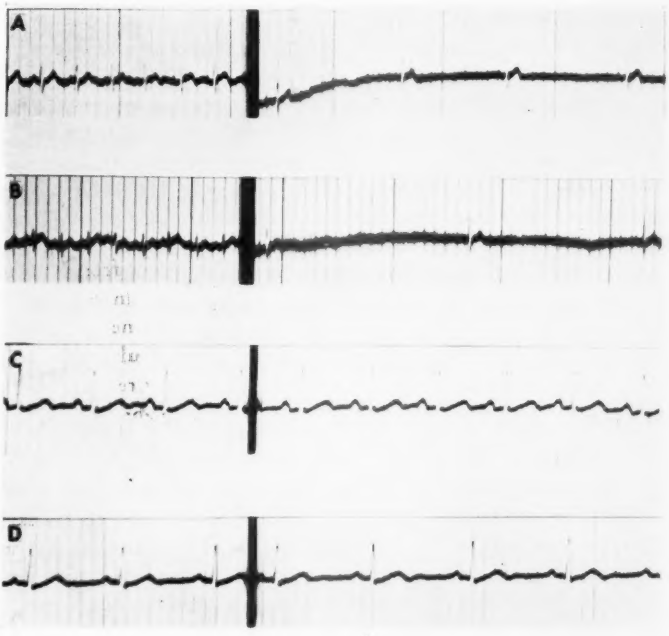

Fig. 8. Electrocardiograms of rabbit and human. Left of broad black vertical lines, heart rate just before application of stimulus. Right of line, rate at height of reflex inhibition. A and B records from rabbit. A. 15 cc. of cigarette smoke diluted with 200 cc. of air; 100 cc. of mixture blown through nose. B. 40 cc. of vapor from ether bottle diluted with 200 cc. of air and 100 cc. of mixture blown through nose. C. and D. Human electrocardiograms. In C, a very large draw (50-60 cc.) of cigarette smoke was expelled through the nose without inhalation or dilution. D. (another subject) painful stimulus of ammonia vapor in nose.

diately expelled, without dilution, through the nose. This was also painful. Neither of these procedures evoked a significant change in cardiac rate, though the concentrations of irritating substances were greater than the maximal concentrations used in the animal experiments. The negative results in healthy human subjects indicate that there are great differences between rabbit and human reflexes, at least in sensitivity.

There are also positive experimental reactions in human subjects which show the presence of a trigemino-vagal cardio-inhibitory mechanism in the human which in certain pathological conditions

exhibits a heightened degree of sensitivity. Russetzki applied stimuli of sinusoidal electrical current to various branches of the trigeminal nerves of about one hundred patients by means of a unipolar electrode on the skin at points overlying the foramina of exit of the branches to a superficial position. The branches stimulated were the supraorbital, infraborbital and mental. Ordinarily the response was a lowering of arterial pressure and slowing of the pulse by 5 to 15 a minute. In some cases, however, the slowing was as great as 30 to 35 a minute or about a 50 per cent diminution from the normal rate. In other cases results were negative and in some an acceleration occurred. Early cases of neuralgia and neuritis of the trigeminal nerve gave large responses. In tabes, results were negative, or in some cases there was acceleration. Other relations of the cardio-inhibitory reflex to disease are described in his paper. The oculo-cardiac reflex (Aschner[1]) is another trigemino-vagal reaction.

Some authors (Dabney,[3] Grove,[6]) have expressed the belief that certain accidents during minor procedures in the nose and sinuses have been due to a trigemino-vagal reflex. According to Killian,[10] this can be true only when the irritability of the trigeminus is much greater than usual, and the human respiratory mucous membrane with normal sensibility does not give rise to severe reflex responses. Our experiments offer confirmation to this statement.

SUMMARY:

Stimulation of the mucosa of the nose, pharynx, and larynx of the rabbit with certain vapors (ether, acetic anhydride, ammonia, cigarette smoke) evokes a reflex inhibition of the heart which bears an S shaped curvilinear relation to concentration of the vapor.

Associated with the cardiac slowing the arterial pressure shows an initial decline in all sensitive unanesthetized or lightly anesthetized animals. The pressure may quickly level off and rise gradually while the heart is slow and show a peak above the original when the heart approaches the normal rate, or pressure may remain below the control level until the original rate is reached.

Touch stimuli in the nose or larynx brings about a fall in pressure which may be very steep with some slowing of the pulse.

Faradic stimuli applied to the nerves supplying the mucosa of the nose, pharynx, and larynx, in general, gives rise to responses which are comparable to those from chemical and tactile stimuli, except that in the case of the trigeminal branches to the mucosa of the nose and skin of the snout the electrical stimuli excite relatively less cardiac inhibition and more rise in pressure and general pain reaction.

The primary vaso-depressor and cardio-inhibitory reflexes are very delicate and may be eliminated by relatively light anesthesia, and they are depressed or eliminated by much surgical and experimental trauma. In an animal in which these are depressed, stimuli, especially in the nose, raise blood pressure, and if the anesthesia is not too deep there is slowing of the heart secondary to the rise in pressure.

Cardiac inhibition, vasomotor depression, vasomotor excitation, and the reactions secondary to increased pressure are each excited by a distinct nervous mechanism.

Human experiments indicate that the cardio-vascular reflex systems of the normal individual are quite insensitive to stimuli in the nose, but studies are cited which show that in certain pathological states irritability is significantly increased.

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XXVIII

THE FACIAL NEURALGIAS: THEIR ETIOLOGY AND TREATMENT

MARK ALBERT GLASER, M.D.

LOS ANGELES

Previous to the studies of Sir Charles Bell[1] in 1829, it was assumed that the seventh nerve was responsible for facial pain rather than the fifth. In the last 110 years tremendous strides have been made in the proper diagnosis and adequate therapy for the relief of facial pain. Not only have diseases of the fifth nerve been held responsible for pains about the face, but lesions of other cranial nerves, peripheral nerves, as well as the autonomic system have proven causative factors. With the advance of medical and surgical therapy, heretofore mutilating and death-dealing surgical procedures have reached such a high state of technical perfection that the mortality of these operations is now as low as four-tenths of one per cent. Furthermore, the proper recognition of these varied and multiple neuralgias has prevented useless surgery upon the teeth, sinuses and even abdomen, for the relief of this pain.

The neuralgias in and about the face and head may be segregated into four main groups: (1) Trigeminal neuralgia; primary and secondary. 2. The otalgias. 3. Other cranial nerve neuralgias. 4. Atypical neuralgia; primary and secondary.

PRIMARY TRIGEMINAL NEURALGIA

Tic douloureux, also called trigeminal neuralgia, chronic paroxysmal neuralgia, epileptiform neuralgia, prosopalgia, trifacial neuralgia, major neuralgia and Fothergill's disease was known to early civilization. Trigeminal neuralgia was recognized by Avicenna in A. D. 1000, and was later described by Schlichtung in 1748; Nicolous Andre' in 1756, who first named it "tic douloureux," and Fothergill in 1773, accurately described the disease.

The pain of tic douloureux, which is probably one of the most severe known to medicine, occurs about equally in men and women. The right side is more frequently involved than the left. The average age of onset is approximately 50 years, though cases have been reported

at 12 and 87 years. It is confined mainly to the second and third nerve divisions, rarely to the first.

The onset is usually sudden and violent. The pain may then leave and the patient be pain-free for months, or even years. After the disease has once established itself, the patient has acute paroxysms of sharp, lancinating, knife-like pains, occurring in attacks of a few seconds, with intervals of freedom. The period of repeated attacks may last from several days to several weeks, and then a remission of months to years may occur. As the time from the original attack increases, the intervals of freedom are lessened and the morale of the patient is lowered so that they are in constant fear of an impending attack.

The attacks are brought on by contact and even draughts of air. Occasionally the only relief from pain is had by going to bed in a dark room and abstaining entirely from food. Trigger zones, or certain sensitive areas exist in the face. Any slight pressure upon them may bring on an attack and are characteristic of the disease.

The pain of trigeminal neuralgia, which is always confined to the sensory distribution of the fifth nerve, never going beyond its bounds, is superficial, and there is absolute freedom from pain between these attacks, although occasionally there is discomfort present in this interval. It is a unilateral disease, primarily, though there have been reported in the literature about fifty-four patients with bilateral pain. Trigeminal neuralgia is a disease of unknown etiology and no single case has ever been cured by the clearing up of oral sepsis, the extraction of carious teeth, the treatment of apical abscesses or sinus operations. In addition, no single case has ever had permanent relief without therapy.

Treatment: Trichlorethylene, a drug discovered by chance in Germany during the World War, I have found of value in 11 per cent of my own series of patients. When a patient is relieved by trichlorethylene therapy, sensory anesthesia does not occur.[2] Alcohol injection of the second and third divisions is only palliative, the period of relief usually continuing from six to eighteen months, and occasionally as long as seven years (Fig. 1). The unfortunate fact is that, after repeated alcohol injections, a period arises wherein further injection of the nerve does not relieve the pain, primarily because a marked fibrosis has occurred about the nerve, thus preventing the infiltration of alcohol. The extra-oral route is utilized by the neurosurgeons, although the intra-oral route of the dental surgeon is of value for the mandibular block. Alcohol should never be injected

Fig. 1. A composite sketch illustrating the point of injection and angle of needle placement in alcohol block of the trigeminal nerve. *A*, Maxillary route of Levy and Baudouin; *B*, Maxillary route of Schlosser; and, *C*, Mandibular route of Levy and Baudouin.

into the first division because of the proximity of the nerves to the eye and the resulting dangers frequently arising therefrom, particularly paralysis of the ocular muscles, and occasionally blindness. I personally do not favor alcohol injection of the ganglion because of the danger of infiltration into the pons along the nerve sheath. Numerous such cases of pontile destruction have been reported in the literature.[3]

The surgical treatment of trigeminal neuralgia has reached the stage of great efficiency.[4] The pain can be absolutely relieved permanently with the minimum risk to the patient and with the surprisingly low mortality of 0.26 per cent. The present accepted operation is that described by Spiller and Frazier,[5] wherein the sensory root is partially sectioned. As the disease rarely involves the ophthalmic division, the sensory supply to the cornea is maintained in order to prevent a keratitis. The operation, which can be per-

formed as readily in the old as in the young, leaves the patient without any facial disfiguration. Intracranial section of the three divisions of the trigeminal nerve, as well as removal of the ganglion, has given way to subtotal section of the sensory root (Fig. 2). Conservation of the motor root is also carried out in order to prevent paralysis of the muscles of mastication and to leave the patient in a position for a bilateral operation, if such is needed[6] (Fig. 3).

In the patients with bilateral trigeminal neuralgia, the posterior route of Dandy is the most efficient.

SECONDARY TRIGEMINAL NEURALGIA

I. *Dental Origin:*

1. Exposure of the dentine, or superficial caries.
2. Unerrupted wisdom teeth.
3. Impacted molars.
4. Dental pulp stones.
5. Impingement of the roots of the molars upon the inferior dental canal.

II. *Tumors:*

1. Gasserian ganglion.
2. Suprasellar brain tumors.
3. Nasopharyngeal.
4. Base of the skull.
5. Root of the fifth nerve in the posterior fossa.
6. Malignant disease about the face and neck.
7. Acoustic neuromas.
8. Cholesteatomas.
9. Osteomas.

III. *Vascular Lesions:*

1. Aneurysms of the internal carotid.
2. Thrombosis of the posterior inferior cerebellar artery.
3. Aneurysms of the basilar artery.
4. Cavernous angiomas surrounding sensory root.
5. Miscellaneous arteries and veins irritating the root of the posterior fossa.
6. Petrosal vein crossing sensory root.

IV. *Inflammations:*

1. Gasserian ganglion.
2. Gradenigo's syndrome.
3. Intra-oral or submaxillary abscess.
4. Cellulitis or Ludwig's angina.
5. Osteomyelitis or periostitis.
6. Adhesions about the sensory root and brain stem.
7. Empyema of the antrum.

V. *Diseases:*

1. Multiple sclerosis.
2. Syphilis.
3. Syringobulbia.

VI. *Traumatic:*

1. Fracture of the skull.
2. Fracture of the jaw.
3. Secondary to dental extraction.

VII. *Miscellaneous:*

1. Myoma cutis.
2. Congenital malformations of the skull.
3. Unknown etiology.

In the secondary group the classification is self-explanatory, showing the various types of pathology which may result in pain within the distribution of the trigeminal zone. The pain in true trigeminal neuralgias is so characteristic that little confusion could exist in its differential diagnosis.[7, 8]

Dandy[9] has reported a series of cases wherein intracranial exploration by the suboccipital route has revealed the presence of vascular and adhesive pathology in and about the sensory root. It is his belief that these abnormalities were responsible for the trigeminal pain. These patients were relieved by subtotal resection of the root. From a clinical standpoint these patients in every way simulated the cases of true trigeminal neuralgia previously described.

There are, however, many extracranial causes of secondary trigeminal pain which irritate the peripheral nerve divisions. Many of these are quite obvious upon examination of the patient, and

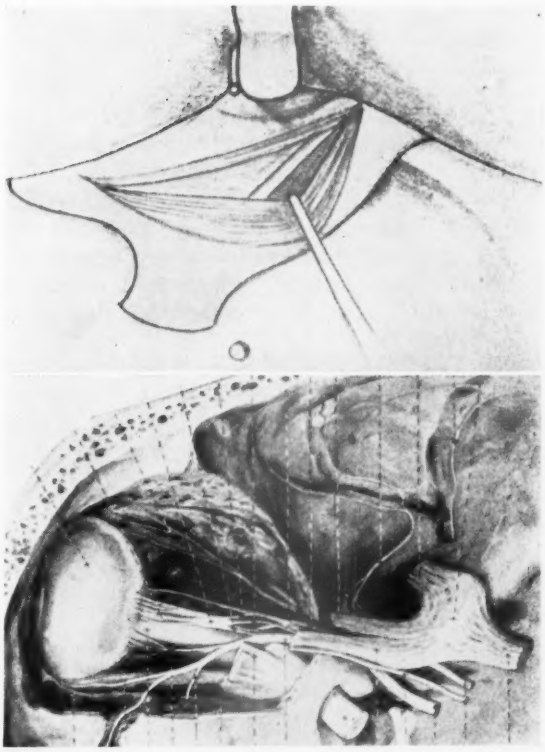

Fig. 2. The modern Spiller-Frazier operation consisting of intracranial subtotal resection of the sensory root, not the ganglion, with preservation of the motor division. The retractor has pulled back the sensory root exposing the motor root, and leaving one-third of the fibers of the root above so as to maintain corneal sensitivity. Compare this to the above anatomical drawing taken from Spalteholz.

therapy must be directed to the eradication of the primary cause. In these particular cases, the pain, as a rule, is of a more chronic nature, and the attacks are longer and less severe. Trigger zones are rare. The pain, however, is of a superficial, cutting, sharp variety, well within the bounds of the trigeminal distribution. The patients may be relieved by alcohol injection, or surgery. In malignancy of the face, injection thereapy is extremely valuable for the relief of the pain, and also facilitates painless surgical measures.

Though the injection by no means cures the primary malignancy, it makes the patient's life one of ease and comfort. Occasionally, we find cases of this chronic nature wherein an etiology is indeterminable, yet relief is secured by alcohol injection.

The sensory supply to the auricle, external auditory meatus and tympanic membrane is extremely complex and is associated with a great amount of overlap. These nerves are the cervical, trigeminal, facial, glossopharyngeal and vagus. This overlap is entirely dependent upon the embryological development. As a result of this sensory supply, primary otalgias, though little described, have, however, been reported in twelve patients (Glaser[10]). From a dental standpoint, reflex otalgia is probably the more important type of ear pain, because it is often secondary to inflammation in the mouth, teeth and jaw. Not infrequently an infected tooth on one side of the jaw refers pain to the opposite ear. These pains will not be relieved by clearing up mouth sepsis, or the extraction of teeth. Except for the cervical neuralgias, these pains occur in attacks of short duration, are acute, lancinating and sharp, are aggravated by irritation of trigger zones, and in every way represent trigeminal neuralgia, except that they follow the respective distribution of the cranial nerve involved. As some of these nerves have a sensory supply to other parts of the head, these patients present even more extensive pictures than otalgias. The otalgias may exist alone, or be associated with wider areas of pain.

Fig. 3. Lateral view of patient following trigeminal surgery. Note the absence of scar.

1. *Cervical Neuralgias:* The second and third cervical nerves supply the area of the head from the trigeminal boundary downward, the mastoid and the anterior and posterior part of the auricle. There is, however, an overlap of sensory supply by the facial and vagus nerves. The neuralgias of these nerves may be divided into two types: (a) the cervico-occipital, and (b) the cervico-otalgic.

A. *The cervico-occipital neuralgia* is rare. Remak has reported 50 cases in a series of 1,500. The etiology is the usual cause of all neuralgias, but may also be produced by arthritis, tumors, injuries

and aneurysms of the vertebral artery. This type of neuralgia must be differentiated from the psychogenic pains so commonly found in this region. The pain follows the bounds of the sensory distribution, is frequently bilateral, and usually paroxysmal in occurrence. The attacks are of longer duration than those of trigeminal pain, and are usually of a more aching character. Occasionally, however, the tenderness may be so acute that ruffling of the hair may start a paroxysm. The head is usually held in a stiff position and during the paroxysms of pain, jerking of the neck musculature with head movements may result. Trophic disturbances, such as graying of the hair or loss of hair may occur.

B. *The cervico-otalgic neuralgias* are extremely infrequent. I have only been able to collect three such cases in the literature. The first case was reported by Nottingham[11] in 1857, and was associated with an acute onset of pain located in the sensory supply of the cervical nerves in the auricle, and closely simulating trigeminal neuralgia, except for the location. The other two cases were reported by Hall[12] in 1932, and myself in 1932. These patients had longer attacks, starting very much like migraine, with a gradual increase of symptoms until a crises was reached. In Hall's case a cervical arthritis was present. In my case the dental hygiene of the patient was probably the worst I have ever encountered. His tonsils were badly infected. Following the clearing up of his dental sepsis and the removal of his tonsils, the pain was entirely relieved.

The treatment of both of these groups must first be directed toward the causative factor. If this does not relieve the pain, injection or section of the cervical nerves can be carried out.

2. *Facial, or Seventh Nerve Neuralgias:* Ramsey Hunt,[13] following studies in herpetic inflammations, wherein pain was localized in the depths of the ear, auditory canal and mastoid region, concluded that the facial nerve is a mixed nerve. He was not certain as to the definite outline of this sensory zone, but as a result of the study of seventeen cases of primary isolated herpes zoster, formed the opinion that this sensory zone consists of the tympanic membrane, the external auditory canal and meatus, the concha, tragus, anti-tragus, anti-helix, fossa of the anti-helix, and lobule of the ear. He did not believe this entire area was represented by the geniculate zone. He felt that allowance must be made for overlapping of the trigeminal in front and the cervical behind, and he further believed that the glossopharyngeal and vagus nerves were also partially represented in this area. Having observed two cases in which the eruption was distributed posteriorly in the cleft between the auricle and the mastoid

process, posterior auricular branch, he assumed that considerable variation existed in the ganglionic representation of the seventh, ninth and tenth nerves in the region of the auricle. The investigations of Retzius, Hiss, von Lenhossek, Penzo, Sappolini, Van Gehuchten and Dixon have shown conclusively that the facial has a sensory ganglion, the geniculate; a sensory root, the pars intermedia of Wrisberg, as well as sensory fibers crossing in the chorda tympani, the great superficial petrosal nerve and the trunk proper of the seventh.

Wilson[14] believes, however, as a result of anatomical study, that there is insufficient evidence present to prove that the geniculate ganglion nerves supply the tympanic membrane. The nerves of the tympanic membrane enter from the external auditory meatus, pass in large trunks from the principal arteries and in small branches along the periphery. These form a plexus in the fibrous tissue from which branches are distributed to a subepithelial and a submucus plexus. In addition, there is to be distinguished a zonular and an intraepithelial plexus. There are also nerves lesser in number that enter from the tympanic cavity. Only one variety of nerve-ending is seen in the epithelium (Fig. 4).

The first clinical case was reported by Max[15] in 1894. At that time he naturally did not realize the nerve involvement and considered the patient a case of otalgia. His method of therapy did not relieve the patient. Clark and Taylor,[16] in 1909, being familiar with the work of Ramsey Hunt, had the opportunity of seeing such a patient who had attacks of acute, lancinating pain, deep in the ear. These pains were of such an excruciating nature that surgery of the seventh nerve was carried out. The seventh, the pars intermedia and the upper fasciculus of the eighth nerve were cut, with an entire relief of symptoms. McLean,[17] in 1935, reported such a patient, who, however, would not submit to surgery, with the consequent continuation of pain. Due to the overlap of sensory fibers of the seventh and ninth nerves, the differential diagnosis from a clinical standpoint in primary deep-seated ear pain is impossible. Reichert[18] has pointed out this important point in the case he had operated upon under local anesthesia, believing he was dealing with a seventh nerve, but in reality he was dealing with a ninth nerve neuralgia. Consequently, operation under local anesthesia with irritation of the nerve is essential to determine which of these two is the offending member.

3. *Glossopharyngeal, or Ninth Nerve Neuralgias:* Weisenburg,[19] in 1910, was first to describe irritative symptoms secondary to the glossopharyngeal nerve. In a patient with a tumor of the cerebellopontil angle, the ninth and part of the tenth nerve were stretched

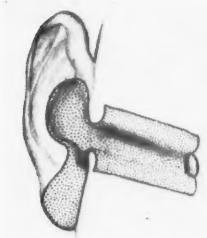

Fig. 4. The facial nerve supplies fibres to the tragus, concha, external auditory canal and tympanic membrane of the ear. These areas are also supplied by the overlap of the trigeminal, glossopharyngeal and vagus.

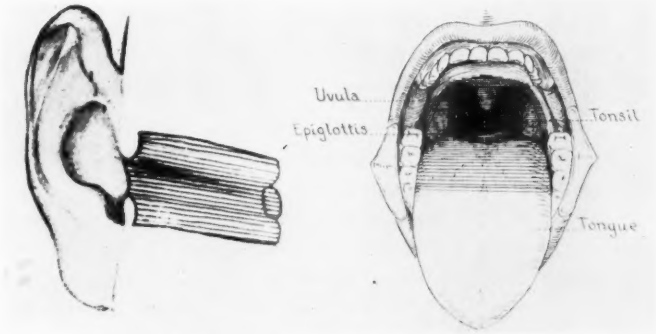

Fig. 5. The glossopharyngeal nerve has a double sensory distribution, thus accounting for the true syndrome of glossopharyngeal neuralgia, and a rather rare syndrome with pain referred to the ear alone.

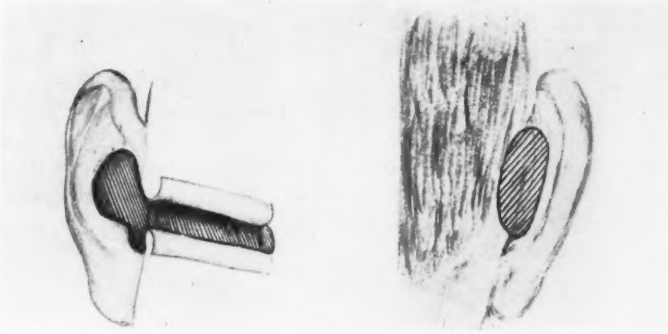

Fig. 6. The sensory supply of the vagus also overlaps the glossopharyngeal, facial, and to a certain degree the trigeminal. The tympanum itself is supplied by fibers from all of these nerves, whereas the concha is supplied by the facial nerve as well.

over the growth, which also compressed the fifth nerve. The pain was only partially relieved when Frazier sectioned the fifth root. This pain was situated in the root of the tongue, referred to the ear, and down the neck; was associated with burning in the throat, a constant sense of dryness, and the paroxysms occurred spontaneously, or were brought on by eating. This Weisenburg attributed to irritation of the glossopharyngeal nerve. In 1920, Sicard and Robineau[20] described three cases met with during the war of what they named "Algie velo-pharyngie essentielle." They did not recognize the nature of this pain, and recommended extracranial section of the glossopharyngeal nerve, the pharyngeal branch of the vagus and the superior cervical sympathetic for the relief of pain. In 1921, Harris[21] reported two cases, which he had followed for a lengthy period of time, and also failed to recognize the clinical picture. Since then Adson,[22] Dandy,[23] Stookey[24] and Doyle[25] have recognized the significance of this pain and have recommended intracranial section of the glossopharyngeal nerve.

The clinical picture may be divided into two groups: the glossopharyngeal neuralgia proper, consisting of pain in the region of the tonsil, throat or base of the tongue, occurring in severe paroxysms similar to those of the trigeminal neuralgia, and radiating to the front of the ear, around the pharynx and palate, the auditory meatus, pinna and back of the lower jaw. The pain occurs spontaneously, or may be brought on by swallowing, eating or by touching the trigger zone at the base of the tongue, or in the tonsillar region. Occasionally the pain may be brought on by touching the pinna. During these attacks of pain, there is a sense of dryness in the throat which is followed by a cough as if the patient was attempting to expel an irritating substance from the throat. In contradistinction to this complete picture, this pain may be confined entirely to the ear and may be confused with the facial nerve neuralgia described by Ramsey Hunt. This neuralgia is primarily concerned with the tympanic plexus branch, and attention has been directed to a patient of this type by Reichert. Other cases of otalgia, secondary to glossopharyngeal neuralgia, have been described by Doyle in 1923, Keith[26] in 1932, Reichert[27] in 1933, Ericksen[28] in 1935, and Lillie and Craig[29] in 1935 (Fig. 5).

4. *Vagus, or Tenth Nerve Neuralgia:* The vagus nerve is distributed to the back of the auricle, inferior posterior wall of the external acoustic meatus and the concha, as well as the tympanic membrane. The superior laryngeal nerve, of which the internal branch is the sensory supply, inervates the mucous membrane of the

larynx, the epiglottis and base of the tongue. Pain deep in the ear has been attributed by Fay to a neuralgia of the vagus nerve, because in cases of malignancy by section of the vagus, he was able to relieve this pain. The pain, however, was not of the paroxysmal nature of the trigeminal attack. Neuralgia of the superior laryngeal nerve has been described by Avellis,[30] Hutter,[31] Bailey,[32] Harris, Echols and Maxwell.[33] It is characterized by paroxysms of unilateral pain, which radiates from the side of the thryoid cartilage to the angle of the jaw, and occasionally to the ear. There is a trigger zone at the plica of the nerve in the pyriform sinus. Swallowing will bring on an attack. There is a sensitive point superficially situated above the lateral to the thyroid cartilage where the nerve pierces the hypothyroid membrane (Fig. 6).

ATYPICAL NEURALGIA

In 1908 to 1916, Sluder[34] wrote a series of articles wherein he described a neuralgia pain based upon involvement of the sphenopalatine ganglion and the sphenoidal sinus. It was quite true that some of these peculiar pains were relieved by therapy directed toward the sphenopalatine ganglion and sphenoidal sinus, but there were a group of other patients who were considered sphenopalatine neuralgias, but whose pain Sluder himself was unable to relieve by this therapy. Pains of this variety were described by Oppenheim, Cushing,[35] Harris and Davis.[36] In 1924, for the first time, Frazier and Russell[37] segregated a group of patients from those with characteristic trigeminal neuralgia, and suggested that because of the peculiar nature of their pains and for want of a better term, these cases be called "atypical neuralgia," in contradistinction to those cases of typical trigeminal neuralgia. In 1928, Glaser[38] segregated 143 of these patients from those of typical trigeminal neuralgia and described the complete syndrome for the first time. In 1938, Glaser and Beerman[39] again described this syndrome, reporting 200 cases in detail.

From a clinical standpoint, these cases have many things in common with true migraine, but in addition, they have some points in common with the cranial nerve neuralgias, particularly the trigeminal. In some instances they so closely simulate trigeminal neuralgia that an alcohol injection is necessary to differentiate one from the other. In the earlier work of Frazier and Russell, and Cushing, a number of these patients were subjected to major surgery on the trigeminal tract, and it was only because of the continuation of pain in spite of the anesthesia that it was realized these patients had some other type of neuralgia. In fact, one may definitely state that a

transitional type exists between migraine and trigeminal neuralgia. Because this transitional neuralgia, which we have classified as "atypical neuralgia," may closely simulate both groups; and further, because many of this group have an associated neurosis, considerable confusion in the nomenclature has existed, with the result that the same clinical syndrome had been given different names by various authors. These names have included psychalgia, facial neuralgium, sympathetic algias, pseudo-trigeminal neuralgia, ciliary neuralgia, migrainous neuralgia, atypical migraine, allergic migraine, sympathetic neuralgias, neuralgic headaches, senile neuralgia, cephalalgia, facial causalgia, cephalalgia of the jaw, sympathetic hemicrania, autonomic facio-cephalic neuralgia, spheno-palatine neuralgia, vidian neuralgia, and psychogenic neuralgia, etc.

In the past ten years some progress has been made in determining the sensory supply of the face, as well as classifying the various clinical entities which could produce the pain of atypical neuralgia. Though this problem still remains one of a perplexing nature, and considerable confusion exists, in this present contribution some attempts will be made to classify the various pain syndromes producing atypical neuralgia, as well as to consider the various phases of present methods of therapy. We no longer need look upon this pain as an incurable condition. However, we must still be ultra-conservative in carrying out therapeutic measures, as has previously been emphasized. We should definitely refrain from any surgical procedures unless we are absolutely certain of our diagnosis. It must still be remembered that incorrectly chosen procedures, such as operations upon the trigeminal tract, surgical removal of the sphenopalatine ganglion, extraction of teeth, operation upon the nose and nasal sinuses, sympathectomies, mastoid and abdominal operations carried out indiscriminately in poorly chosen cases, will do far more harm than good, and leaves the patient in a much worse condition than previous to surgery. I cannot to urgently advise caution in arriving at a decision for operative measures.

These neuralgias may be classified into four main groups:[10] *Group I* consists of the primary atypical neuralgias for which, at present, no cause can be ascertained, and for which the treatment must be directed toward the blockage of sensory pathways. This diagnosis is only reached by a process of elimination. Without doubt, in the future we shall be able to remove from this group a great many neuralgias of definitely known etiology. In *Group II* a series of clinical entities give rise to the pain of an atypical nature. Treatment naturally must be directed towards the disturbances. However, the

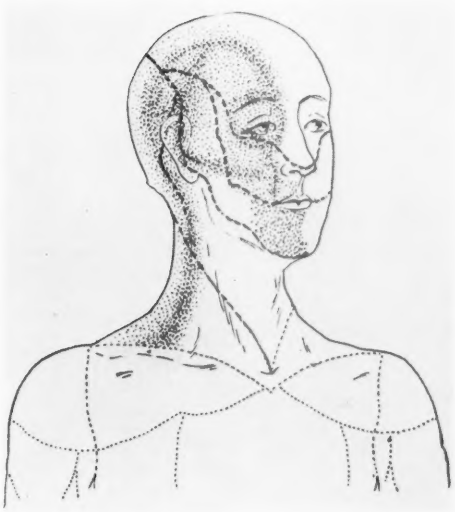

Fig. 7. The dashed lines represent the trigeminal distribution in the face and the cervical distribution in the neck. The dotted shaded area represents complete distribution of the atypical pain. Note how it crosses the bounds of the trigeminal distribution in the face, the cervical nerves in the neck, and the facial, glossopharyngeal and vagus nerves in the ear. This area, to a certain degree, follows the route of the large blood vessels.

blockage of nerves may be essentially necessary for the relief of pain. *Group III* contains patients in whom generalized systemic diseases are responsible for the pain. In *Group IV* associated pathology of either the head, neck, or abdomen exists and the neuralgia disappears in most cases with the clearing up of such abnormalities. In all of these groups, if the neuralgias do not clear up, specific treatments directed toward the autonomic system in the form of medication, or surgery, should be utilized. It is well to mention here that though this classification and the therapeutic suggestions might indicate that the entire problem of atypical neuralgia has been solved, we shall still be confronted with a group of unfortunate patients whom we cannot relieve of their pain. Thus, the problem continues to remain one for further research, with the hope that eventually all these patients can be assured of complete relief from this troublesome, annoying, painful neuralgia.

CLASSIFICATION OF ATYPICAL NEURALGIA

I. *Primary Atypical Neuralgia.*

II. *Clinical Entities Manifesting Atypical Neuralgia:*

1. Sphenopalatine and vidian neuralgia.
2. Postherpetic trigeminal neuralgia.
3. Trigeminal ghosts.
4. Trigeminal ghosts with lingual spasm.
5. Mandibular joint pathology.
6. Autonomic facio-cephalalgia.
7. Painful tic convulsif.
8. Hypertonic neck muscles.
9. Senile neuralgia.

III. *Systemic Diseases Producing Atypical Neuralgia:*

1. Allergy.
2. Endocrine disturbances.
3. Psychoneurosis.

IV. *Pathology in the Head, Chest and Abdomen Manifesting Atypical Neuralgia:*

1. Infections about the head.
   a. Mastoiditis.
   b. Cavernous and longitudinal sinus thrombosis.
   c. Deep-seated facial abscesses.
2. Tumors of the head and neck.
3. Intracranial pathology.
4. Dental pathology.
5. Nasal septum deviations and spurs.
6. Ocular pathology.
7. Chest pathology.
8. Abdominal pathology.
7. Pelvic pathology.

PRIMARY ATYPICAL NEURALGIA

The clinical picture of primary atypical neuralgia has previously been discussed in detail and differs distinctly from the acute neuralgias secondary to trigeminal facial, glossopharyngeal, vagus and cervical nerves. The primary group of atypical neuralgias has had one distinct point of differentiation from the secondary group. All these cases of primary atypical neuralgia, which have come under my observation, and in which great difficulty in the relief of pain had

Fig. 8. A patient during a spasm of Painful Tic Convulsif.

existed, were cases wherein the pain had been present for a year or more. This pain is never superficial, is always deep-seated, of an aching, burning or throbbing type. The patients find great difficulty in accurately describing the sensation. From their accounts of pain, I have assembled a list of some seventy different descriptive terms, such as: dull, aching, throbbing, burning, shooting, sharp, sense of pressure, drawing, needle-like, boring, toothache, pulling, soreness, gnawing, bursting, tearing, electricity, hot iron, tingling, smarting, nagging, knife-like, beating, stinging, pricking, bugs creeping, itching, gripping, lightning. In addition, the following descriptions have been used: the sensation was like tearing celery; a thousand fishhooks pulling and tugging on the face; birds flying under the skin; the pounding of a hammer on the face; a hot poker back of the eye; the cutting away of pieces of bone or muscle beneath the skin; a bruise; a saw cutting the face; something in the jaw; a mass of fire; pins and needles; stiffness; the feeling of menthol; the buzzing of a mosquito; a log on top of the head; a full feeling; a hard knot deep in the eye; the bursting of an eyeball; the pushing of the eye through

the head; drawing out the eye, a ball of fire or electricity in the eye; twitching, severe jumpy, crawling, unbearable, swearing, pounding, surging, crushing, vibrating, excruciating or grabbing.

In the majority of this series of 250 cases no cause could be attributed to the onset of pain, though numerous coincidental events were thought by the patients to be responsible for their difficulties. Thirty-eight felt that pain followed the extraction of teeth; sixteen remembered the onset of pain shortly after an accident; whereas, thirteen believed the pain followed surgery; six had numerous minor causes as the etiology of the complaint. It is quite apparent from this study that no particular factor could be held responsible for the onset of this pain, and the events named were coincidental, excepting possibly the extraction of teeth. In tic douloureux the sexes were equally affected; whereas, in atypical neuralgia, females predominate. Bilateral pain occurs in only two per cent of the cases of tic douloureux, while it is found in 33 per cent in the cases of atypical neuralgia.

The distribution of the pain in trigeminal neuralgia was always in the areas supplied by the trigeminal nerve, whereas in atypical neuralgia a circular area within the vascular supply of the face and head was affected. The pain was felt in the chin, along the nose, around the eye, over the brow, to the vertex or temporal region, in front of, in, or through the ear; and thence down into the suboccipital region. Occasionally it entered the shoulder, rarely the body. This wide area of distribution could occur in a single case or there might be individual areas, or various combinations of areas (Fig. 7).

The pain of trigeminal neuralgia is brought on by the slightest local contact, and trigger zones occur over the facial foramin. In the atypical group there was occasional tenderness over the cervical sympathetic ganglion, or the carotid artery. The pain of atypical neuralgia is aggravated by such local factors as cold, draft, heat, eating, light, contact, brushing teeth, vibrating, reading, wind, blowing the nose, sneezing, swallowing, shaving; and such general factors as: fatigue, excitement, menses, worry, talking, winter, exertion, night, lying down, stooping, morning, noise, dampness, motion, cough, arguments and constipation.

Sympathetic phenomena are present in about 50 per cent of the cases of atypical neuralgia, but never present in trigeminal neuralgia. These sympathetic phenomena consist of ocular disturbance, such as lacrimation, edema, corneal injection, unequal pupils, blurred vision, photophobia, enophthalmos. In addition, the following symptoms,

some of which are common in migraine, have been present: nausea, vomiting, flushing of face, nasal discharge, perspiration, salivation, puffy face, feeling of warmth, ringing in ears, soreness over temporal artery, chills, aural discharge.

Outstandingly different from trigeminal neuralgia is the continuous nature of this deep-seated, aggravating pain without periods of relief. As has been previously mentioned, trigeminal neuralgia manifests itself by short, momentary attacks of excessively severe pain. The atypical patient has a continuous pain of a more annoying, aggravating nature, disturbing by its persistence and chronicity, rather than its severity. Superimposed upon this chronic pain the majority of these patients complained of attacks of greater or less severity, which would come on either acutely or insidiously. If an attack of pain had an insidious onset, it would gradually increase in intensity over a period of from several hours to several days. Then the pain would be at its height, over a period ranging in length from several hours to several days. During this period of most severe pain associated sympathetic phenomena were present in some cases, and the patient would necessarily be confined to bed. The attack might then either suddenly subside or gradually disappear. These attacks might occur at intervals of several days to several months, but during the interim between attacks the patient was never free from pain. This phase of the disease picture demonstrates its close alliance to migraine, and an even closer alliance to migraine is demonstrated by a small number of patients who have frequent attacks, as above described, with only a minor persistent ache, or even at times no ache in the interval between attacks. Another small group of these cases is more closely allied to the trigeminal type, in that these attacks may last for a half hour to several hours, and occur rather frequently in the day during a susceptible period. However, upon close questioning, it is found that the character of the pain differs from that of the true trigeminal group. At times, however, one cannot differentiate this picture from clinical trigeminal neuralgia, and an alcohol injection is used as a therapeutic test. Narcotic addiction is common in the atypical group, but never among the trigeminal patients.

Most of these patients have an associated neurosis, which has led to the belief of many that the entire group are psychoneurotics. However, one can readily believe the development of a psychoneurosis secondary to this persistent, annoying, aggravating pain from which no relief can be secured. This would seem closer to the truth than to consider the whole picture one of a nervous patient. It is true, of course, that some of these casese are definitely psychoneu-

rotics, but they will be considered, in detail, under the secondary types of atypical neuralgia.

SECONDARY ATYPICAL NEURALGIA

In this series of 250 patients with atypical neuralgia, 143 belong to the group reported in Philadelphia. In none of these patients were we able to relieve the pain. In the first fifty of the second group of patients observed, the pain was only relieved in about 10 per cent of the instances. In the second twenty-five patients observed, secondary causes were found in approximately 65 per cent of the patients, and about a 50 per cent relief of symptoms. In the last twenty-seven an etiology of the pain was determined in 85 per cent of the cases, and 75 per cent were relieved of their symptoms. Of interest is the fact that most of these patients had their pain for only a short period of time, from several days to three months, in contradistinction to those of the primary cause wherein the pain was present for a year or more before they came under by observation. From a clinical standpoint, the best curative results were obtained in those cases wherein the pain was of a short duration.

The treatment of atypical neuralgia must first be directed to the more conservative methods, and as a primary atypical neuralgia in most instances is diagnosed by a process of elimination, the various forms of medicinal therapy should first be utilized. Among those of value are trichlorethylen, ergotamine tartrate, adrenalin, amyl nitrate, esserine, atropine, and tamponades of borate of glycerin in the ear, and the choline compounds. In addition, the use of mild sedatives of the barbiturate group may be used in conjunction with these drugs. Further therapy consists of cocainization of the sphenopalatine ganglion, removal of the properly selected infected teeth, or poorly constructed bridges, occasional septal operations with the removal of spurs, and rarely sinus surgery.

In cases of hypertonic muscles of the neck, diathermy and removal of foci infection are indicated, while electric stimulation and mirror practice is of value in cases of painful tic convulsif (Fig. 8). Mandibular joint pathology must be treated by reposition of the jaw, by the interposition of the discs between the molar teeth, or more radical surgery directed toward the mandible. In cases of postherpetic trigeminal neuralgia, nerve block of the trigeminal, occasionally operation upon the trigeminal, and attacks upon the autonomic system may be of value. Trigeminal ghosts offer considerable difficulty and may be relieved in certain instances by alcohol injections of the stellate ganglion, or surgery upon the autonomic system.

Associated lingual spasm, which is a rare occurrence, may be alleviated by section of the lingual nerve in the mouth. In cases of systemic diseases, such as allergy, endocrine disturbances, or psychoneurosis, treatment must be directed toward the specific diseases. In the more acute cases, associated with infection or tumors, operative intervention is indicated. Ocular pathology must be taken care of immediately and in many instances gives surprising results. Secondary pain resulting from chest, abdominal and pelvic pathology must be alleviated by treatment of these particular areas. It is important to know that this pathology, in many instances, may account for the atypical pain.

Should all of these methods prove of little or no avail, the treatment must be directed toward the autonomic system. First, in the form of nerve block, either alcohol or novocain as a test, or later the more radical surgical procedures. The surgery that has relieved these patients has consisted of excision of the inferior and upper two thoracic ganglia with decortication of the carotid artery. Occasionally fifth nerve procedures must be utilized in conjunction with sympathetic surgery. Removal of the stellate ganglion, the superior cervical ganglion and the plexus upon the vertebral artery has been carried out for the relief of this pain.

From this rather brief discussion, it is quite apparent considerable headway has been made in the last ten years for the relief of pain in these patients. Each case requires an exacting study and analysis. Conservative measures should first be utilized, and at no time should useless surgical procedures be attempted without certainty of their success. It must be remembered, as has been pointed out, that in many instances the more that is done for these patients, the worse they become.

CONCLUSIONS

1. Trigeminal neuralgia is one of the most favorable pains to relieve because of the certainty of the cure following section of the nerve. Temporary relief may be obtained by alcohol injection, or, in certain selected cases, inhalation of trichlorethylene.

2. The neuralgias of the cervical, facial, glossopharyngeal and vagus nerves may be relieved by alcohol injection, or actual section of the nerve.

3. Atypical neuralgia is closely allied to migraine and has been described in the literature under a varied nomenclature. For many years these patients have been considered a hopeless therapeutic prob-

lem. We should no longer be baffled in our attempts to remedy these pains. One cannot overemphasize the care that should be taken in properly selecting the procedure in each case. Radical measures should be the last resort, and every conservative means should be exhausted before resorting to the extraction of a tooth. The problem, at present, is by no means solved, but definite progress has been made in eliminating the pain in a great number of cases; some 75 per cent have been relieved in the last twenty-seven patients under observation.

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XXIX

PATHOLOGY OF THE TONSIL WITH STATISTICAL REPORT AND MICROSCOPIC STUDY*

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During the past few years many reports and studies have been made on the general pathology of the tonsil. This extensive literature, I believe, indicates an increased interest on the part of the profession in the diseased tonsil. There is no organ of the body perhaps that has received more attention from the profession than the tonsil, in so far as it is concerned with focal infection and its effect upon the general system. We read and hear much discussion on what constitutes a diseased tonsil, particularly in regard to chronic inflammatory change. The incidence of tuberculosis, tumors, trichina, and other more rare conditions has been well covered in the literature. In order to put more emphasis on the importance of more careful study of diseased tonsils and at the same time point out some of the more interesting pathology found in routine examination, I would like to present the following study.

Every tonsil received in the laboratory was fixed in formaldehyde, then sectioned longitudinally at right angles to the mucous surfaces, dehydrated in acetone and embedded in paraffin. Sections were made and stained with hematoxylin and eosin.

During the last sixteen years 12,370 pairs of tonsils have been examined in the laboratory of St. Joseph's Mercy Hospital, Sioux City, Iowa. In 1926, I reported[1] on 3,845 pairs of tonsils studied microscopically up to that date, and made a special study of syphilis of the tonsil. Since then, up to December 1, 1938, 8,516 additional pairs of tonsils have been studied.

In this group seven cases of tonsilar tuberculosis, five cases of syphilis of the tonsil, four cases of tumor (two malignant and two benign), and one case of trichinosis were found. The remaining

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cases all showed in varying degree evidence of acute or chronic inflammatory change.

With one exception, in the cases of tuberculosis no evidence of clinical active tuberculosis was found. In the one case some clinical and x-ray evidence of active tuberculosis was found subsequent to tonsillectomy. From this study we learn that care should be exercised in making a diagnosis of acute tuberculosis elsewhere in the body, and certainly the patient should not be given a bad prognosis. A thorough check should be made, however, to determine if the patient possibly has any evidence of active tuberculosis.

In the first group studied, 0.282 per cent of cases showed active lesions of the tonsil. In the second group only 0.082 per cent showed active lesions. There was a decrease in the second group. It would seem that tonsillar tuberculosis is on the decrease. I believe the two groups are comparable, for in both groups the cases come from the population of Sioux City and the surrounding territory.

Magee,[2] of the University of Michigan, reviewed a series of tonsillectomies and compared them with Weller's previous group. He found a definite decrease in tonsillar tuberculosis. The incidence for the years 1933 to 1935 being 0.44 per cent as compared with 2.35 per cent for the years 1906 to 1919.

Syphilis of the tonsil is not as frequent as tuberculosis. The five cases in the second group occurred in adults. Of the four cases reported on the first series, one was congenital in a child and the others acquired syphilis in adults. All these adult cases represent a late stage of acquired syphilis. The common pathological lesion found was a diffuse epitheloid proliferation with many giant cells. New blood vessel formation was present in all and served to differentiate the lesions from a diffuse tuberculosis of the tonsil. Grossly, the tonsils showed nothing definitely pathognomonic of syphilis. Three showed some hypertrophy, none were enormously enlarged. No mucous patches were present. The tonsils showed some increase in the consistency and some were somewhat friable. In all cases both tonsils were involved, though usually not to the same extent. The lesions were deep in the lymphoid tissue, away from the crypts, and occasionally the germ centers showed the only lesions. In all cases subsequent Wassermann reactions were positive. The lesions were tertiary in nature in all cases and were not associated with the mucous lesions of secondaries. Usually many spirochetes could be demonstrated.

Trichina spirals in the tonsils is a rather rare finding in my experience. Only one case was found. The parasites were found only in the muscle tissue, found in and attached to the capsule. Both tonsils were involved. The patient came in because of muscular rheumatism and tonsillectomy was advised, because of the rheumatism. General muscular tenderness with stiffness and pains about the muscles of the neck and chest should always arouse suspicion of trichinosis. A differential blood count may give considerable aid in diagnosis. In this one case the eosinophiles formed 56 per cent of the differential. The patient followed a stormy period of several months and finally recovered. In histological examination of tonsils, occasional calcified areas will be noted in the capsules and in the muscles attached thereto. Such an area, I believe, means an old calcified trichina spiral.

Of the four tumor cases, two were malignant and two benign. One was a lymphosarcoma in a boy 16 years of age, the other a squamous cell carcinoma in an adult. The benign tumors were both papillomas in adults.

The inflammatory changes of the tonsils represented a wide variety and considerable time should be spent on this phase, as it holds most interest for the clinician and the surgeon. Diagnosis of acute or chronic change depended on several findings. A diagnosis of acute inflammation was made when the mucosa of the tonsil showed an exudate in the crypts as well as on the surface. This may be a fibrino-purulent exudate in the form of a membrane. The crypts were filled with fibrin and pus cells and many times red blood cells were present. There were varying amounts of edema and polynuclear infiltration in epithelium and lymphoid tissue. Occasional small focal abscesses were found. They may be central and away from the crypts. They therefore must be metastatic in the lymphatics or blood stream and not cryptic in origin.

Chronic changes are less apparent. A tonsil shows chronic change if on examination scar tissue is found; the capsule is thickened. There is an increase in the small blood vessels and even angioblastic proliferation may be seen. Cartilage and even bone tissue may be found in the capsule and large trabeculae. While cartilage and bone represent, without a doubt, a metaplasia of connective tissue, yet this process is incited by chronic inflammation. Cryptic changes are most interesting. Here surface infection can come in close proximity to, if not in immediate contact, with lymphatic drainage of the tonsils. The tonsil is a lymphoid organ and its location makes it a submucous organ. The lymphatics originate in the tonsils. The

lymph supply is efferent only[3] and no lymph vessels lead to the tonsils. This fact is evident—any involvement of the nasal sinuses or pharyngeal wall never invades the tonsils. Metastatic tumors in these regions never secondarily involve the tonsils. Even carbon or insoluble dyes injected in the mucosa of the throat, nose or sinuses will reach the cervical lymph glands but will not appear in the tonsils. For this reason cryptic infections are very important. In the crypt various changes occur. In the first place ulceration may occur, leaving contact with the open lymph spaces and lymphoid tissue. However, I do not think that ulceration is the most common change in the epithelium of the crypts. Lymphocytic infiltration of the cryptic epithelium is more or less normal within certain limits. Chronic infection causes a marked increase in this infiltrative process of the epithelium; even the crypts may contain many lymphocytes, as this infiltrative process increases the epithelium atrophies, desquamates, and becomes more or less replaced with lymphocytes. A single layer of epithelial cells can be seen lining the crypts, and often only a few rudimentary epithelial pegs will remain. This means in the end a direct connection between the infected crypt and the lymphoid tissue with all its possible absorption and lymphatic involvement. Here, I believe, is the explanation why the cervical lymph glands may show involvement before the patient is aware of any local trouble in the tonsils. Later tonsillitis is apparent, but the lymph glands become involved early. This also explains the continued long standing or chronic involvement of the cervical lymph glands. Until the crypts become epithelized and a barrier set up between the tonsil and the crypt infection, rapid absorption continues. Finally epithelization takes place and protection is set up.

A word about the removal of tonsils with diathermia. In my experience in microscopic examination, I have yet to see a tonsil so treated to show any evidence of being removed. The volume of lymphoid tissue is as large as found in any average tonsil. The crypts are still present, many of which are plugged and show marked retention of secretions and desquamation of epithelium. Even the ducts from the mucous glands in the capsule and subcapsullar tissue are closed and markedly dilated. I am very skeptical that any operator could be skillful enough to destroy the lymphoid tissues to the capsule and yet not get through into the deep structures of the throat. These tonsils after treatment are more dangerous as a focus of infection than before attempted removal.

In the prodromal stage of measles, interesting histological changes may be found in the tonsils. Warthin[4] was the first to de-

scribe these changes. Here, as shown in Fig. 16, many multi-nucleated giant cells are found scattered throughout the tonsils. The germ centers show the more marked reaction. Occasionally small areas of focal necrosis could be seen also. These areas contained a few polynuclear leucocytes along with cell fragments. The great majority of these even occurred within the germ centers, though they might be located any place in the tonsil. There was considerable edema and congestion. The giant cells may contain from two to fifteen nuclei in large, round or globoid congregations. They seem to form in the germ centers and migrate elsewhere and even may appear in the mucosa. The endothelium of the blood vessels also showed marked hypertrophy; sometimes the endothelium would appear to fill the entire lumen of the blood vessel.

CONCLUSIONS

In the routine histological examination of 8,516 additional pairs of tonsils, seven cases of tonsillar tuberculosis, five cases of syphilis of the tonsils, four cases of tumor of the tonsil (two malignant and two benign), and one case of trichina were found.

There was a marked decrease in the incidence of tonsillar tuberculosis over the first group.

Chronic changes are present in varying degree in all tonsils. The relation of crypt infection, whether acute or chronic, to lymphatic involvement is discussed.

Diathermia does not offer an adequate means of removal of tonsils in the hands of the average operator.

During the prodromal stage of measles the essential pathology is the occurrence of multi-nucleated giant cells in the tonsils.

Pathological findings in the tonsils justify routine examination.

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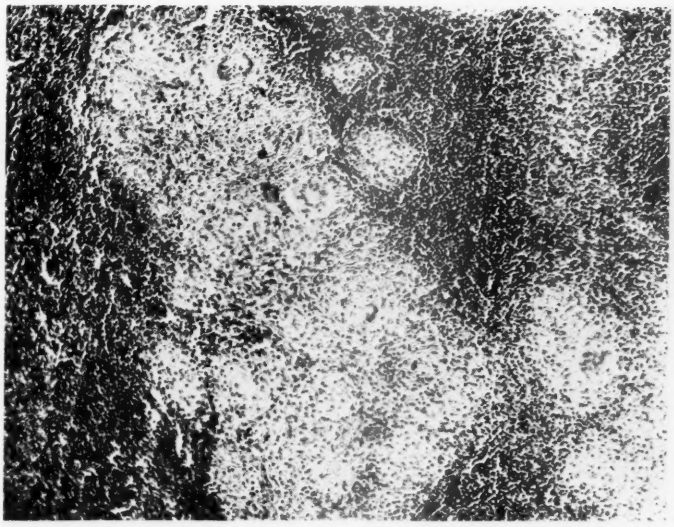

Fig. 1. Photomicrograph of tuberculous lesion of tonsil, showing epitheloid tubercle with many typical giant cells.

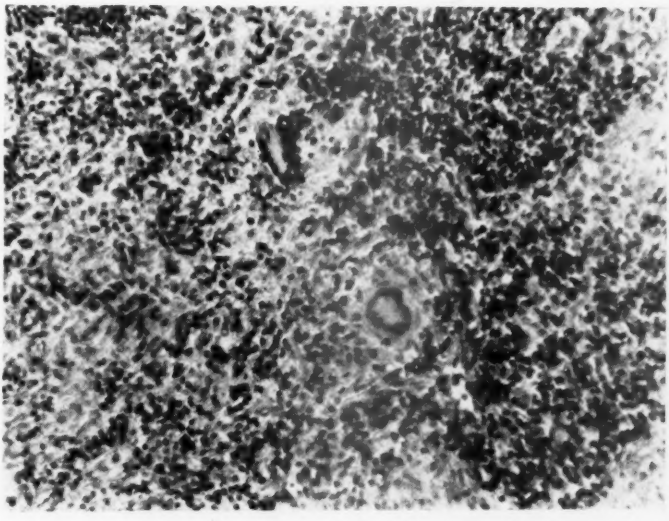

Fig. 2. Section from syphilitic tonsil, showing marked degree of epitheloid proliferation with occasional giant cell.

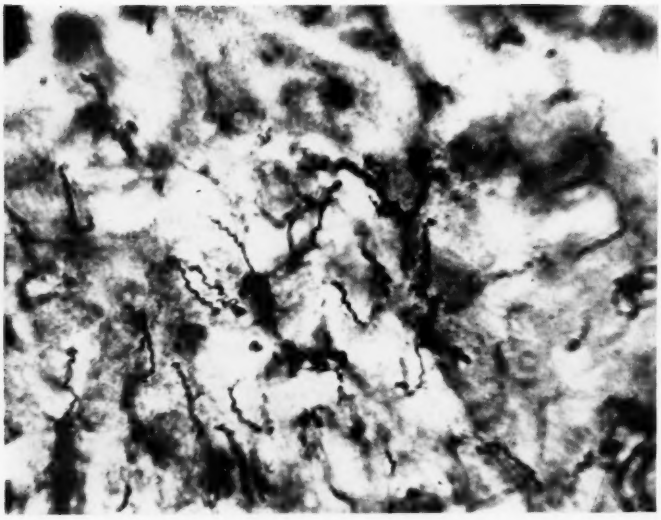

Fig. 3. Photomicrograph of Warthin Starry silver stain, showing numerous spirochetaepallidae in tonsil lesion.

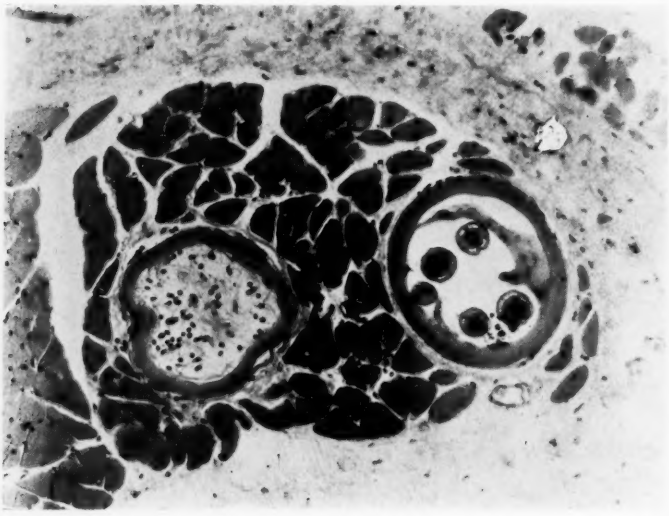

Fig. 4. Section of tonsil, showing two incysted trichinae spirals in muscle bundles attached to capsule of tonsil.

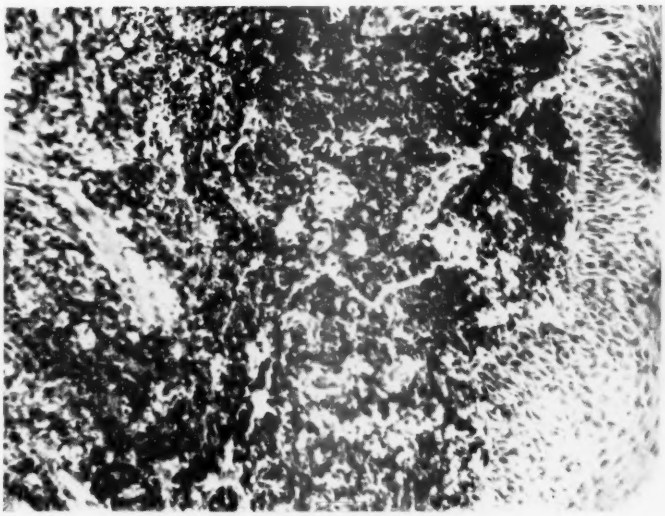

Fig. 5. Lymphosarcoma in 16-year-old boy.

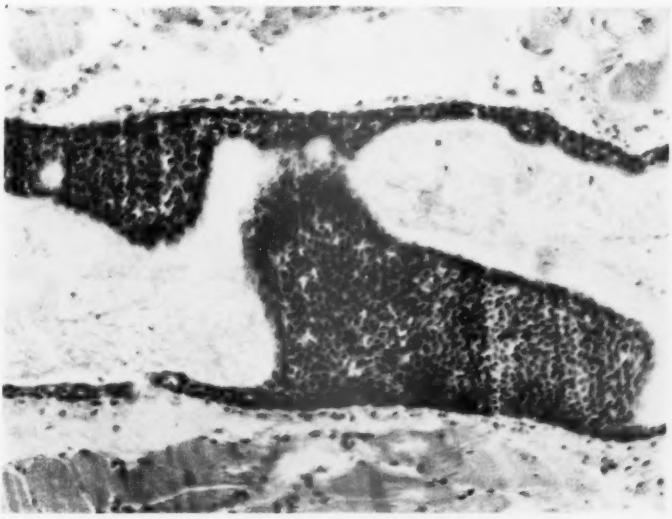

Fig. 6. Section showing an epithelial tumor developing in a duct connecting the mucous glands in the capsule with a crypt in the tonsil.

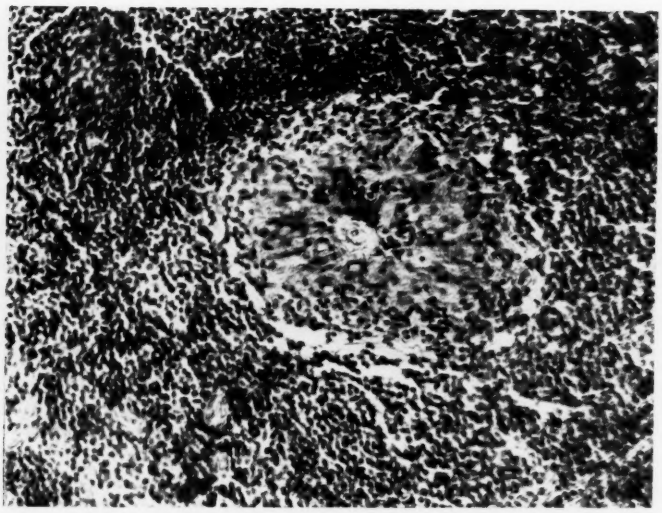

Fig. 7. Section showing a foreign body tubercle in tonsil. The foreign body can be seen centrally located.

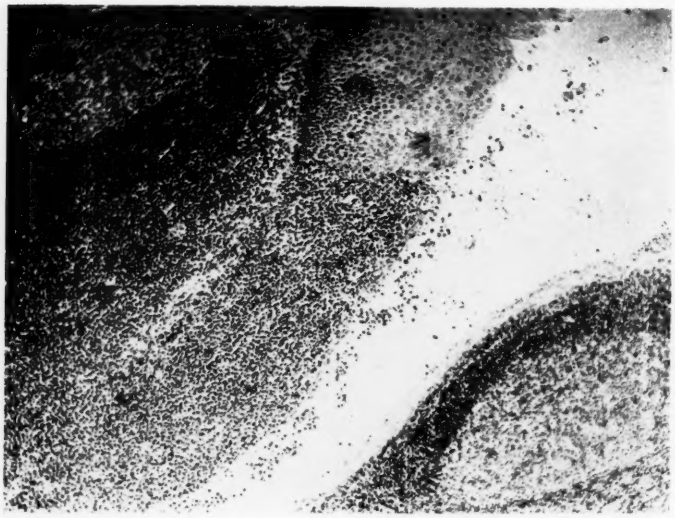

Fig. 8. Section of tonsil showing the abrupt displacement of the crypt epithelium with lymphoid tissue. Drainage from the cysts into the lymphatics is unobstructed.

Fig. 9. Section of tonsil crypt lined with epithelium, showing marked hyperkeratosis.

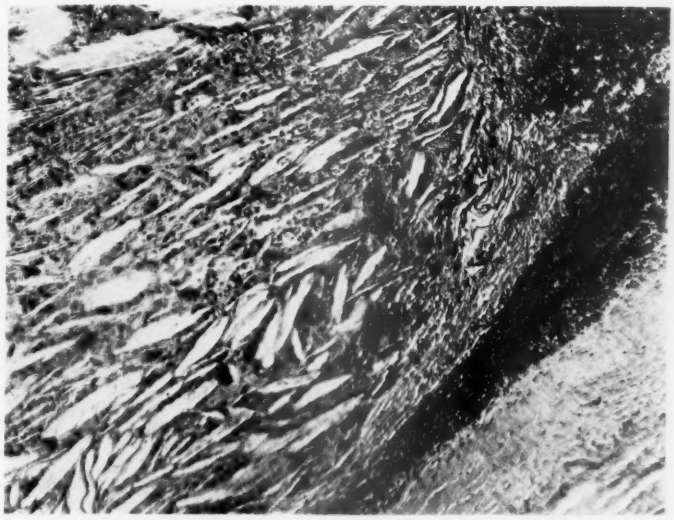

Fig. 10. Section showing an obstructed crypt filled with desquamated epithelium, keratohyalin, leucocytic and cholesterin crystals. No definite epithelial lining is present.

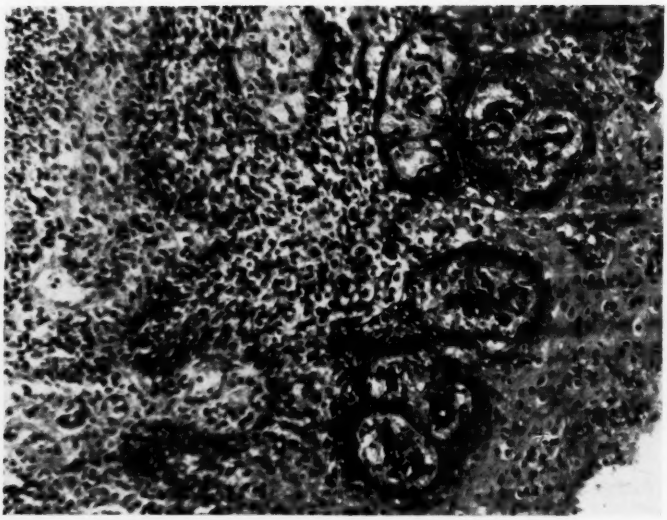

Fig. 11. Section of tonsil showing the numerous lymphatics of the papillae in the crypt epithelium. The lymphatics are filled with deposits of reduced silver.

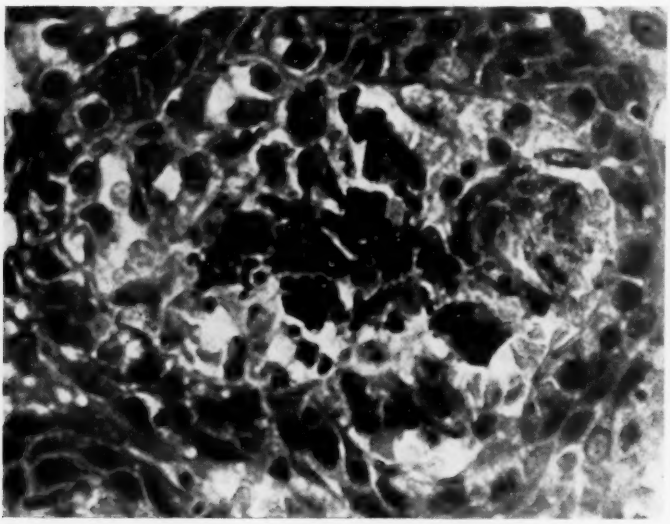

Fig. 12. High power of one of the papillae shown in Fig. 11.

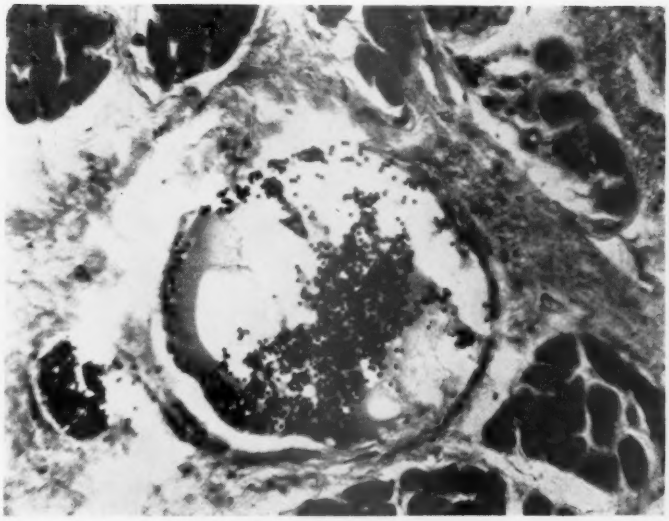

Fig. 13. Section showing one of the large collecting lymph vessels in the capsule of the tonsil.

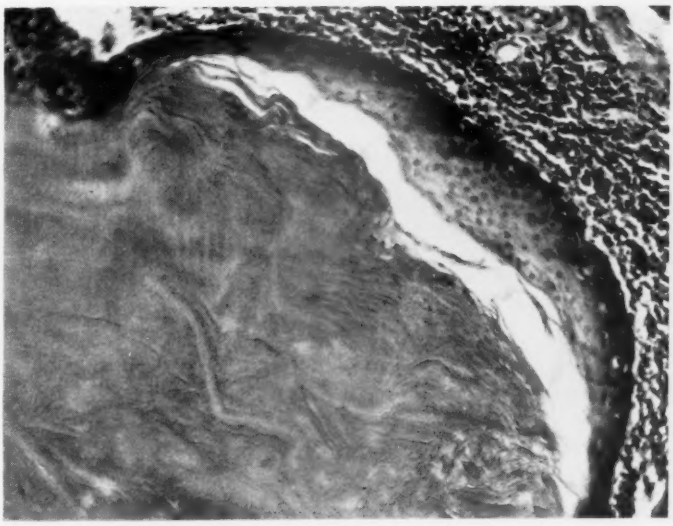

Fig. 14. Tonsil showing a large plugged crypt. Tonsil removed after diathermia.

Fig. 15. Cartilage and bone formation in capsule of tonsil.

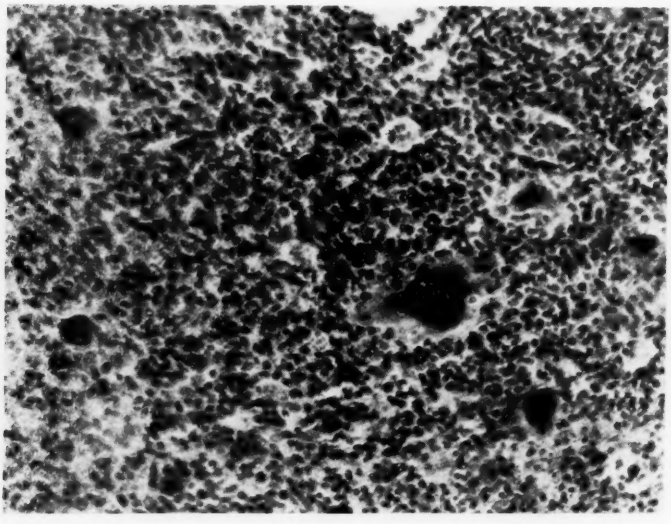

Fig. 16. Section of tonsil showing typical giant cell formation in the prodromal stage of measles.

XXX

ALLERGY IN OTOLARYNGOLOGY AND ITS RELATION TO OTHER MANIFESTATIONS*

II. Diagnosis and Treatment with Case Reports

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In the management of allergy of the nose and paranasal sinuses a detailed clinical history and physical examination are of primary importance. With this data all information obtained from laboratory procedures should be correlated.

When a patient consults the otolaryngologist with such common complaints as sneezing, itching of the nose, nasal obstruction, nasal discharge, or local pain and headache, first consideration is usually given to the question of whether the condition is acute or chronic. Among the acute or subacute conditions, one usually suspects the common cold with or without involvement of the paranasal sinuses, an acute exacerbation of nasal allergy or the onset of hay fever. Among the chronic conditions such affections as deflection of the nasal septum with obstruction, hypertrophic rhinitis of adolescence, perennial nasal allergy, nasal polyps or chronic infection of the paranasal sinuses, must be considered.

On the basis of the nasal symptoms, the changes in the nasal mucosa and the demonstration of eosinophiles in the nasal secretions, the diagnosis of nasal allergy is readily established. Further investigation of the sinuses by means of the x-ray and bacteriologic studies complete the local examination. With the presence of complicating infection, it is important at this point to determine whether it is acute, subacute, or chronic. These various factors in the nasal investigation have already been discussed in Part I.

In the beginning, treatment of the nose and paranasal sinuses should be restricted to local and palliative measures, deferring all operative procedures until the allergic investigation has been completed and allergic therapy established.

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*Presented at the Midwestern Section Meeting of the American Laryngological, Rhinological and Otological Society, January 26, 1938, at St. Louis, Missouri.

In order to institute allergic therapy on a rational basis, it is necessary, first, to complete the clinical history, the physical examination and the laboratory procedures.

The clinical history must be thorough and complete. It must consider every possible relationship of the symptoms to the etiologic factors and to the possible association of other manifestations of allergy.

First, the family history is important. In about 70 per cent or more of all cases there is a history of the occurrence of various manifestations of allergy in the family.

Second, the association of other manifestations of allergy in the past and present history must be analyzed. This association is found in approximately 70 to 75 per cent of all cases of nasal allergy.

Third, the nasal symptoms and associated manifestations should be thoroughly considered from the following standpoints:

Onset—Date, month, year.

Sudden or gradual.
Residence or occupation at time.
Change of environment.
New furniture, clothing, or other articles.
Unusual circumstances or contacts.
Unusual drugs or foods.
Pollen seasons.
Other manifestations of allergy.
Acute infections—cold, LaGrippe, acute infectious diseases or other diseases.
Infancy, childhood, puberty, pregnancy, menopause.

Course—symptoms constant or intermittent.

Analysis of acute exacerbations or periods of relief.

Vacation, business or pleasure trip.
Change of occupation.
Change of residence.
Hospital admission.
Food, drugs, serums, etc.

Relation of attacks to:

Season—spring, summer, fall, winter.

Pollens and seasonal foods.
Changes of residence or occupation.
Unusual inhalant contact.

Time of day.

Environment—home.

Bedroom, living room, kitchen, basement.

Pillows, mattress, bed coverings, draperies, rugs, rug paddings, overstuffed furniture, cosmetics, and toilet articles.

Plants, insecticides. Dogs, cats, birds, toys.

Neighborhood.

Grasses, weeds and trees.

Factories, stables, etc.

Sports—inhalant contact.

Automobile—cushions and upholstery.

Occupation—inhalant contact.

Theatre or other locations.

Foods—dislikes, disagreements.

Symptoms caused by foods—nasal, bronchial, headache, G. I., skin, G. U., etc.

Climate—temperature, humidity, wind, sunlight, altitude.

Physical agents—heat, cold, light.

Acute respiratory infections and acute infectious diseases.

Nose and throat and other operations, treatments. Drugs, etc.

Other diseases or illnesses.

As emphasized by Rackemann,[2] exact dates are important in associating symptoms with possible contacts with offending substances. Exact dates are likewise valuable in determining the reason for the relief of symptoms.

Having completed the history and physical examination, the skin tests are next in order. It is important to emphasize that skin tests are not infallible. Positive skin reactions may occur in the absence of clinical manifestations. On the other hand, clinical sensitiveness may occur even though the skin tests are entirely negative. Of all the antigens used in skin tests, the pollens, however, are the most reliable. In any event, the information obtained from skin testing should be correlated with clinical history and other laboratory procedures.

Upon completion of the clinical investigation, nasal allergy and other associated manifestations may be classified as follows:

I. Seasonal occurence of nasal symptoms.

1. Hay fever.
   a. Tree.
   b. Grass.
   c. Weed.
2. Non-seasonal or perennial.
3. Seasonal (Pollinosis) and non-seasonal or perennial.

II. Associated manifestations of allergy. (Seasonal or perennial.)

1. Allergic bronchitis.
2. Bronchial asthma.
3. Gastrointestinal allergy.
4. Urogenital allergy.
5. Allergic headache.
6. Skin allergy.

   Eczema, urticaria, angioneurotic edema, purpura, contact dermatitis.

On the basis of the clinical investigation and the skin tests, one or more of the following antigens or agents represent the cause of symptoms:

1. Inhalant—pollen, animal danders, dusts, etc.
2. Ingestants—foods, beverages, drugs, etc.
3. Injectants—sera, drugs, vaccines, hormones, allergens, insect bites.
4. Bacteria, molds, parasites.
5. Physical agents.
6. Nonspecific and secondary factors.

Having determined the causative factors, treatment is based upon their avoidance or elimination. Offending foods, drugs and other ingestants should be eliminated from the diet. Orris root, animals, feathers and other agents may be removed from the environment. Sometimes it is necessary for the patient to change his environment or occupation in order to avoid contact with offending inhalants. In all cases prophylactic measures should be instituted to decrease or eliminate contact with common inhalants such as feathers, animal emanations, dusts, etc.

When prophylactic measures fail, it is usually possible to give satisfactory relief by hyposensitization measures. A series of subcutaneous injections to such agents as pollens, house dust, occupa-

tional dusts or specific inhalants may be administered. The injection of food extracts for hyposensitization has not proved satisfactory except in certain specific instances. Tolerance to offending foods may be established, however, by oral administration of small amounts in increasing doses. In complicated cases it may be necessary to institute a program of prophylactic measures, hyposensitization injections and food avoidance.

In the beginning, the treatment of the nose and paranasal sinuses should be confined to local and palliative measures, until symptoms have been controlled by allergic therapy. Local treatment of the nose is usually not well tolerated. As a rule, more satisfactory relief can be obtained by the oral administration of ephedrine or related compounds.

By means of local inspection and the correlation of cytologic studies of nasal and sinus secretions, bacteriologic and x-ray examinations, an accurate estimation of the existing conditions can be determined. The pathologic state of the nose and sinuses, therefore, may be classified as follows:

1. Gross nasal changes—permanent, transitory, residual.
   a. Pallor or simple edema (acute or chronic).
   b. Red type.
   c. Chronic edematous thickening—prepolypoid.
   d. Polyposis—acute or chronic.
2. Gross paranasal sinus changes. Permanent, transitory, residual.
   a. Simple edema—acute or chronic.
   b. Chronic edematous thickening—prepolypoid.
   c. Polyposis—acute or chronic.
3. Complications.
   a. Acute infection.
   b. Subacute infection.
   c. Chronic infection.

Upon improvement of the allergic symptoms there is a tendency to resorption of the edematous changes. Simple edema and edematous thickenings may diseappear. It is always a question whether the edematous process is transitory, permanent or residual. Acute edema is likely to be temporary or transitory. Chronic edema may be of

the fixed type with little tendency to change. Residual changes are those which remain after the allergic process has become quiescent. Such residual edemas are not infrequently observed at the end of the hay fever season or in chronic cases in which the symptoms have become quiescent spontaneously or as the result of allergic therapy. Polyposis may show a marked decrease in extent under allergic management. Reduction may vary from 100 per cent to no change whatsoever. It is not unusual, however, to note a decrease of 75, 50 to 25 per cent. In any event, surgical procedures on the nose and sinuses must be deferred until these changes can be observed. If surgical interference is then necessary, more satisfactory results can be obtained if the allergic process has been controlled. The recurrence of postoperative and subsequent polyposis can be eliminated to a marked extent.

When infection is superimposed upon the allergic process in the paranasal sinuses, it should be definitely determined whether it is acute, subacute, or chronic. This can be accomplished only by repeated and continued observations of the cytology of the secretions, the bacteriologic and x-ray findings.

The persistence of marked polypoid changes with cystic degeneration of the sinus mucosa and the presence of chronic infection are definite indications for surgical interference. The patients subjected to surgical procedures on the sinuses should be most carefully selected. If allergic management has failed to improve the pathologic conditions, two possibilities exist. Allergic methods of treatment may fail because the pathologic changes are permanent or allergic methods have been inadequate and the offending factors have not been controlled. The more skillful and successful allergic therapy is instituted, therefore, the fewer the patients subjected to radical surgery. In any event, if the allergic reactions are not controlled the symptoms may continue and the pathologic changes may recur, in spite of well-performed radical surgery.

The following cases illustrate some of the important problems confronted in the management of allergy of the nose and paranasal sinuses with or without the association of other manifestations. As a routine procedure all patients were instructed to encase the pillows and mattresses for protection from the dust contained therein. The use of washable bed coverings of cotton or wool was advised. Orris-containing soaps and cosmetics, insecticides, and household pets were eliminated when suspected as offending factors. Instructions for keeping house dust at a minimum were also given.

Perennial nasal allergy may persist for many years without complicating infection, the development of polyposis or the association of other manifestations of allergy, as illustrated in the following case:

REPORT OF CASES

CASE 1.—*Nasal Allergy.*—Mrs. E. G., age 54 years, reported for observation for the first time on September 7, 1938, complaining of attacks of sneezing, nasal obstruction and discharge. The nasal symptoms began about 20 years ago without apparent cause, and gradually became more pronounced until they were continuous.

Several years ago the inferior turbinates were cauterized with no relief. One year ago a wheat, egg and milk-free type of diet was tried with no success. The use of special pillows and mattress and orris-free cosmetics was likewise followed by no relief. Scratch and intracutaneous tests showed no reactions. The patient has been employed in a real estate office and does very little housework. There was no history of other manifestations of allergy.

The examination of the nose showed a pale, boggy mucosa with practically complete obstruction and loss of the sense of smell. There was a constant profuse discharge of clear, mucoid secretion which showed many eosinophiles upon microscopic examination.

A complete set of skin tests showed no reactions to pollens, other inhalants, and foods. There was no reaction to house dust. Although this reaction was negative, the administration of injections of dust extract was given on empiric basis. There was an improvement in symptoms following the first injection. After several of these treatments were given, the patient was about 90 per cent relieved of symptoms. Nasal respiration was restored and the sense of smell returned.

Cases of this type with negative skin reactions, especially in older patients, are not uncommon. In the absence of other manifestations and the failure of diet trial, inhalants appeared to be the most logical etiologic factors. Injections of house dust extract were therefore instituted and most satisfactory results were obtained.

In some cases, especially those which have not been controlled by allergic management, nasal polyps are likely to recur after removal. The following case illustrates this point:

CASE 2.—*Nasal Allergy with Nasal Polyps.*—Dr. W. W., age 26 years, was seen for the first time in December, 1934, complaining of nasal obstruction, discharge and sneezing. His mother had hay fever and nasal polyps. The patient's symptoms began in the spring of 1933, while living in Montana. In the fall of 1934 he came to St. Louis. In December, 1934, I removed several large nasal polyps from each side of the nose. No allergic therapy was instituted at that time. During the summer of 1935, while in Montana, the nasal obstruction became marked and the nasal polyps were again removed while there. He had satisfactory relief until December, 1936, when I saw him again with both sides of the nose filled with polyps.

At this time the nasal polyps were removed only from the left side of the nose. Within a period of two weeks there was a return of acute edematous polyposis on the left side. Skin tests showed no positive reactions except a small one to house dust.

Dust therapy was immediately begun, and within two or three weeks the acute edematous polyposis on the left side showed a marked decrease. About six weeks later the left side of the nose was completely free of polyposis and the extensive polyposis in the right side, which had not been disturbed by surgical procedures, showed marked shrinkage. At the end of three months both sides of the nose were free of polyps, with a return of nasal respiration and the sense of smell. After three years of dust therapy there has been no recurrence of symptoms or reapperance of the polyposis.

Recurring polyposis of this type presents a very distressing problem to the otolaryngologist, because the relief following the removal of the polyps is only of short duration. With the control of allergic reactions, however, prolonged or even permanent satisfactory relief can be expected in a large percentage of these cases. On the other hand, superimposed infection in addition to the polyposis may result in pathologic changes which require radical surgical interference.

In hay fever, such as the ragweed type, for example, the patient may be sensitive only to pollen. In many cases, however, additional sensitivity may be present to other inhalants, such as dusts and molds, as well as foods. In order to obtain satisfactory results in these cases, treatment must also take into consideration this extra-pollen sensitivity. It is also very important in hay fever patients to determine whether nasal or other allergic manifestations are active outside the hay fever season. Sneezing, obstruction and discharge, cough, or so-called frequent colds may be the symptoms of nonseasonal nasal allergy. Diagnosis can be readily established by cytologic studies of the nasal secretions for eosinophiles.

The following case illustrates the importance of extra-pollen sensitivity and the occurrence of nonseasonal symptoms:

CASE 3.—*Hay Fever and Perennial Allergy.*—Miss E. M., age 27 years, was seen for the first time on October 19, 1927, complaining of nasal obstruction, postnasal discharge, and occasional attacks of sneezing. She gave a family history of allergy in that a paternal uncle had nasal allergy. There was a past history of urticaria as a child.

The nasal symptoms began in the summer of 1925 as grass and ragweed hay fever, and have recurred every year since that time. In the fall of 1936, the nasal symptoms became perennial but were rather mild and consisted principally of nasal obstruction and postnasal discharge. No other manifestations of allergy have appeared, except nausea from the ingestion of egg.

The examination of the nose on October 19, 1937, showed a pale, boggy mucosa with scanty clear secretion which showed many eosinophiles. Skin te-

by the cut or scratch method showed positive reactions to the following pollens: giant ragweed, short ragweed, marsh elder, Russian thistle and Bermuda grass. Positive reactions were also noted to the molds alternaria, hormodendrum and monilia sitophilia. By the intracutaneous method positive reactions occurred to: chicken, duck and goose feathers, orris root, pyrethrum, stock house dust, and egg.

By the avoidance of egg, feathers and orris contact, combined with the subcutaneous injections of dust extract, the nonseasonal symptoms practically disappeared.

During the summer of 1938 a series of preseasonal injections of grass and ragweed extract, combined with the injection of dust and mold extracts, resulted in very satisfactory control of the hay fever symptoms.

Satisfactory control of the hay fever in this patient was dependent upon the administration of dust and mold extracts, the avoidance of orris and feather contact, and the elimination of egg from the diet, as well as the injection of pollen extracts. The nonseasonal symptoms were mild and intermittent and might have been easily overlooked. The dust injections were continued throughout the year.

In children, colic, urticaria or eczema may occur in infancy, disappear by the age of two years, and never reappear. On the other hand, nasal symptoms and asthma may follow the infantile allergy. Allergic bronchitis or asthma in children not infrequently follow the acute infectious diseases, especially pertussis. Nasal allergy with cough and mild asthma may occur throughout childhood or continue into adult life without recognition of the allergic cause of the symptoms. These patients are often treated for colds and bronchitis, and even subjected to tonsillectomy without relief.

In the following case the patient's symptoms of nasal allergy, cough and mild wheezing began in childhood and continued into adult life.

CASE 4.—*Nasal Allergy and Allergic Bronchitis.*—Mr. E. F. S., age 35 years, complained of nasal obstruction and postnasal discharge with occasional attacks of sneezing. He also complained of cough, which was considered the most annoying symptom.

His father had hay fever and asthma. The patient's nasal symptoms and cough began in childhood. The cough was confined mostly to the winter months. As the nasal symptoms consisted mostly of obstruction and postnasal discharge, the allergic nature of the complaint appeared indefinite. There were no other manifestations of allergy except a tightness in the chest which occurred with attacks of severe coughing.

The examination of the nose showed a boggy type of mucosa but with no definite pallor. The secretions were rather thick, tenaceous and mucoid. The

microscopic examination showed a large number of eosinophiles and a few neutrophiles.

A series of skin tests to the common antigens showed no reactions except a small positive wheal to house dust.

The patient was instructed to avoid feather and wool contact in the bedding. A series of injections of house dust were instituted. Marked improvement followed the first injection. With subsequent injections the cough and tightness in the chest disappeared. The nasal symptoms subsided and free nasal respiration was restored.

This patient had no other allergy besides the respiratory manifestations and his symptoms were almost entirely confined to the winter months, suggesting that house dust was the etiologic factor. The response to the first injection of dust extract was dramatic. He has continued to be free of symptoms with this type of therapy.

Some patients with nasal allergy and asthma may have the bronchial symptoms only upon occasional contact with certain inhalants or foods. Acute colds may likewise precipitate attacks of asthma. The following patient had more or less constant nasal symptoms but the asthma occurred only on specific occasions.

CASE 5.—*Nasal Allergy and Bronchial Asthma.*—Miss M. J., age 27, was seen for the first time on September 21, 1937, complaining of nasal symptoms of obstruction, discharge, sneezing, and occasional attacks of bronchial asthma.

There was a positive family history of allergy, in that the father had bronchial asthma. The patient stated that she had hay fever from June until October every year between the ages of nine and fifteen years. Since childhood, however, the patient has had frequnt colds and some mild nasal symptoms all the time.

In December, 1936, following pneumonia, the first attack of bronchial asthma occurred. Recurring attacks persisted for about four weeks. During the subsequent five months there was no further asthma, but it reappeared in June, July and August, 1937, in mild form. On September 17, 1937, a marked attack of asthma occurred which necessitated hospitalization for five days. There was no history of other manifestations of allergy in addition to the respiratory types.

The patient has been employed by a publishing concern where there is contact with paper and glue, but these substances do not appear to be significant factors.

The examination of the nose showed a pale, boggy mucosa with thin, clear secretions which contained many eosinophiles. The chest was clear.

Skin tests by the intracutaneous method showed borderline reactions to feathers, pork, rye, rice, sweet potato and egg; definite positive reactions were noted to wool, ozite, horse dander, dog dander and house dust.

The patient was instructed to use orris-free cosmetics and to enclose the mattress and pillows in dust-proof encasings. House dust extract has been administered by subcutaneous injections. The patient has shown a marked improvement in the nasal symptoms and has had no asthma except on two occasions. An

attack was once precipitated following an effort to clean a closet and on another occasion upon contact with cat hair. On two occasions mild asthma followed a slight overdose of dust extract.

When asthma follows pneumonia it is always a question of whether the pneumonia was an allergic process or an infection. Asthma, however, may follow either condition. On the other hand, the patient may be entirely free of asthma for a long period of time following a pneumonia of the infectious type. This point is illustrated in the following case:

CASE 6.—*Nasal Allergy, Asthma and Allergic Headache.*—J. R. V., a white man, aged 20, was seen for the first time on September 13, 1937, on account of nasal allergy, asthma, headaches and drowsiness. He gave a family history of hay fever, but no history of infantile allergy. Onset of asthma at the age of three years followed whooping cough. At the onset he was worse in winter than in summer. He had pneumonia in November, 1933, which was preceded by severe asthma for two months. Following the pneumonia he was free of asthma for two years. The asthma returned in the fall of 1935. Nasal symptoms had been more or less constant, but had never been very marked. Since the fall of 1935, he had had intermittent asthma. For two years he had had almost constant headache and a sense of fullness in the head. The headache was described as a general feeling of pressure, especially in the vertex region. The headache was often associated with mental confusion and dizziness.

Examination of the nose showed a pale, typically allergic mucosa. The tonsils were cleanly removed. Ears, pharynx and larynx were negative. The nasal secretions contained many eosinophiles. The patient, therefore, presented a case of nasal allergy and asthma complicated by headache and possibly angioneurotic edema.

Skin tests by the cut method showed definite positive reactions to alternaria and monilia sitophilia molds. Intracutaneous tests showed positive reactions to pyrethrum, horse dander, stock house dust, and such foods as oats, rye, navy beans and milk. The leucopenic indices showed incompatible curves for wheat, rye, rice, corn, but a compatible curve for oatmeal. Definite incompatibility was also shown for egg, beef and tomato juice. Compatible curves were obtained for pea and potato. Following the injection of an extract of stock house dust along with an extract of alternaria and monilia sitophilia, there was marked improvement of the asthma. With increasing doses of the above, the asthma completely subsided. After the completion of the skin tests and the leucopenic indices, the patient was placed on an elimination type of diet which gave complete relief of the headache.

Although this patient had complete relief of asthma for two years following pneumonia, his dyspnea and wheezing began originally following an attack of pertussis. In children, the first manifestations of allergy may appear in infancy as urticaria, eczema, or colic. Later, nasal symptoms may follow. Then following pertussis, asthma may appear for the first time. In a series of 200 cases of nasal allergy and asthma in children, we[2] have made an analysis of the relation of asthma to the acute infectious diseases. This data was

available in only 133 of the cases. In 56 cases pertussis preceded the onset of asthma and in 13, or 23.3 per cent, it was definitely associated with the cause of the onset of the asthma. Measles preceded the onset of asthma in 67 cases and was associated with the onset of asthma in nine cases, or 13.4 per cent. In 17 instances scarlet fever preceded the asthma and its association was definite in three cases, or 17.7 per cent. In 17 cases of pneumonia the onset of asthma occurred in five, or 29.4 per cent. In three cases of diphtheria, the onset of asthma occurred in two, or 66.6 per cent. Among the 133 patients on whom we had records of the occurrence of acute infectious diseases, 104 had one or more of these diseases before the onset of the asthma, and in 32 or 30.8 per cent, the infectious disease was definitely associated with the precipitation of asthma.

Although an individual may be affected with allergy from infancy or childhood into adult life, there is in general a tendency to shifting from one manifestation to another. Infantile allergy may persist, disappear, or reappear. Childhood and adult manifestations may do likewise. Gastrointestinal or skin allergy or allergic headache may have been present before or at the time of the onset of respiratory manifestations. They may disappear or persist with the respiratory symptoms. Later, new manifestations may develop after the onset of respiratory allergy. The following case with multiple manifestations illustrates these points:

CASE 7.—*Nasal Allergy, Bronchial Asthma, Gastrointestinal Allergy and Allergic Headache.*—Mrs. G. B., age 31, came under observation for the first time on March 17, 1938, complaining of nasal allergy, bronchial asthma, and gastrointestinal symptoms.

As a child she had gastrointestinal symptoms, frequent colds and bronchitis. Later, the nasal symptoms and bronchitis subsided, but she continued to have some gastrointestinal symptoms, off and on up until the time of observation. Between the ages of 18 and 25 she was subject to attacks of severe headache. In March, 1935, a gastrointestinal x-ray showed a diverticulum in the second portion of the duodenum, and adhesions about the cecum probably resulting from an appendectomy performed in 1921. The conditions were alleviated by an abdominal operation. Skin tests and an x-ray of the chest made in 1936 were negative.

The nasal symptoms of discharge, obstruction and sneezing began in February, 1936, following an acute rhinitis. The nasal symptoms continued intermittently until June, 1936, when cough and wheezing began. In November, 1936, bilateral Caldwell-Luc operations were performed. The cough and asthma disappeared for a period of about three weeks. A change to a warm, dry climate in January and February, 1937, resulted in a marked improvement, but a return to a cold northern climate again precipitated the respiratory symptoms. In May, 1937, bilateral ethmoidectomy was performed with no relief of symptoms. A change to a warm, dry climate where she remained from June to October, 1937, again was

accompanied by satisfactory relief. A return to the northern climate again resulted in cough and asthma.

When the patient was first observed she was able to sleep only two or three hours each night. Tightness in the chest was constantly present and the cough was most persistent. The patient would often start to cough and wheeze before completing a meal. There was constant nasal obstruction and profuse discharge. She also complained of a sense of soreness in the upper abdomen with cramps and gas. There was no apparent relation between specific food ingestion and the symptoms.

The nasal examination showed a pale, boggy mucosa with edematous polypoid tissue into the middle meatus. There was a profuse discharge of clear mucus from both the nose and bronchi. Many eosinophiles were present in the secretion from both sources.

Intracutaneous skin tests to the common foods and inhalants showed no definite positive reactions except to house dust. The patient had been taking injections of adrenalin every day for some time previously; therefore, poor skin reactivity was expected. Leucopenic indices showed incompatible reactions to wheat, corn, rice, rye, orange, egg and potato. Compatible reactions were noted for milk, banana, potato, oats and tomato. The patient was placed on a diet free of the above incompatible foods, including nuts, spices, condiments and chocolate. By diet trial it was noted that cough and asthma followed the ingestion of wheat, prunes, pineapple, coffee, peanuts or cherries. During the period of dietary manipulation the patient was also given injections of house dust extract at weekly intervals. About six weeks after the beginning of the treatment the patient was free of nasal symptoms, cough, asthma and gastrointestinal disturbances.

This patient had various manifestations of allergy beginning in childhood and persisting into adult life. Although well performed radical surgical procedures were done on the nose and paranasal sinuses, the respiratory symptoms and the pathologic changes in the nose and sinuses continued. Satisfactory relief was obtained only after the institution of allergic management. The etiologic factors consisted of both inhalants and foods.

Although nasal allergy may have its onset in the wake of a severe acute cold, LaGrippe, an acute infectious disease, puberty, pregnancy, the menopause, or more specific disease, as well as exposure to an overdose of some inhalant, food, or other antigen, it may also begin with or immediately follow some other manifestation of allergy.

In the following case, nasal allergy developed immediately in the wake of an acute attack of urticaria and angioneurotic edema.

CASE 8.—*Urticaria, Angioneurotic Edema and Nasal Allergy.*—H. E., age 19, reported for the first time on July 7, 1936, with an acute keratitis in the left eye of two weeks duration. He had no complaints referable to the nose, throat and ears. The keratitis cleared up within two weeks under local treatment.

On November 18, 1936, the patient returned with an acute generalized urticaria of seven days duration which came on without any apparent cause. A few days later he also developed a generalized edema of the angioneurotic type. Finally, he also had joint pains and swelling, such as that frequently noted in serum disease.

Skin tests performed by the cut or scratch method showed a one-plus reaction to pineapple and rye, two-plus reactions to green pepper, squash, raisins and horse dander, and three-plus reactions to asparagus, banana, white potato, milk, pork and salmon. On a modified wheat, egg and milk-free diet the urticaria and angioneurotic edema with the joint symptoms disappeared within a period of two weeks.

A few days later the patient reported for observation, complaining of sneezing, nasal obstruction and discharge from the nose. An examination revealed a pale, boggy type of mucosa with clear mucoid secretion. Microscopic examination of the secretions showed a large number of eosinophiles, indicating that the patient had developed a nasal allergy for the first time. The patient did not return for further observation, so the final outcome was not determined.

Upon the development of nasal symptoms, this patient was under the impression that he had an acute cold. The examination of the nasal secretions, however, revealed a marked eosinophilia.

In the following case, nasal allergy and asthma also appeared in the wake of a severe urticaria, but the patient had had other manifestations of allergy for many years previously.

CASE 9.—*Nasal Allergy, Asthma, Gastrointestinal Allergy, Allergic Headache, Urticaria and Angioneurotic Edema.*—Mrs. M. DeR., age 43, was observed for the first time on November 12, 1938, on account of nasal obstruction, sneezing and discharge. The nasal symptoms began in early September, 1938, following a prolonged attack of urticaria. The nasal symptoms were very pronounced, as the nose was completely obstructed to breathing most of the time and the discharge was very profuse. About November 1, 1937, an otolaryngologist removed several polyps from the right side of the nose.

This case is particularly interesting from the standpoint of the past history of other manifestations of allergy. At the age of 18 years the patient had an attack of asthma following the ingestion of aspirin. Nine years ago, in 1938, during the postoperative period of a pelvic operation, aspirin was administered for headache. An attack of asthma was precipitated. Previous to the pelvic operation she had had severe attacks of headache for seven years, but they did not reoccur for a while after the operation. During the past nine years, however, she has been subject to attacks of indigestion, manifested by the symptoms of gas, distension and upper abdominal cramps. She also suffered with fatigue and occasional headaches. She occasionally noted marked swelling of the lips and face. Fluctuations in the body weight of six to eight pounds were also noted. An increase in weight occurred coincidentally with abdominal symptoms.

In May, 1937, marked generalized urticaria appeared and persisted until August, 1937. The nasal symptoms began for the first time immediately following the subsidence of the urticaria.

The examination of the nose revealed a very boggy, edematous mucosa with small polyps in each middle meatus. The secretions were clear and mucoid. Microscopic examination showed many eosinophiles.

Skin tests by the intracutaneous method to the common pollens, inhalants and foods showed borderline reactions to house dust, feathers, rye, salmon, black pepper and apple. No definite positive reactions were elicited. The leucopenic index showed incompatible curves to wheat, corn, rice, potato and tomato. Compatible curves were obtained to oats, rye, egg and milk. The nasal symptoms subsided immediately upon following a restricted diet avoiding wheat, corn, rice, potato, tomato, beans, chocolate, nuts, spices and condiments. By further diet trial it was found that oranges, chicken, turkey, beans, beets and pineapple caused gastrointestintal symptoms; chocolate caused headache. Upon removal of all offending foods from the diet, the patient became free of all symptoms.

In this case, it is interesting that acute edematous nasal polyps developed within two months after the onset of nasal symptoms. They disappeared, however, after the control of the allergic reactions. In the past history, it was significant that an individual attack of asthma was precipitated by the ingestion of aspirin. It is also significant that this patient suffered for many years preceding the onset of respiratory symptoms with gastrointestinal allergy, angioneurotic edema and allergic headache. Finally, the nasal symptoms began after a severe and prolonged attack of urticaria. The etiologic factors proved to be foods, and the patient was most satisfactorily relieved of all manifestations by specific food avoidance.

COMMENT

The diagnosis and treatment of allergy of the nose and paranasal sinuses, with or without associated manifestations, are dependent upon a thorough and complete clinical and laboratory investigation. The value of these studies has also been emphasized in previous publications.[3, 4, 5]

From the otolaryngological standpoint a great deal of stress must be placed upon the necessity of complete studies of the nose and paranasal sinuses. In the first place, a diagnosis is important. Atypical cases should not be overlooked. Then, the pathologic state of the sinuses must be studied with the idea of determining the type and extent of involvement in each individual sinus. Cytologic, bacteriologic, x-ray and histopathologic information must be carefully correlated with the clinical picture. Local treatment should be primarily conservative except in unusual cases. Radical procedures should be deferred until allergic methods of treatment have been instituted. In any event, satisfactory relief of symptoms and the

control of pathologic changes are dependent upon adequate allergic management.

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XXXI

PROPAGATIONS OF INFLAMMATORY PROCESSES BETWEEN PETROUS BONE AND ENDOCRANIUM OF "FRUSTRATED" FORM*

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Material for the study of the conditions involved in the extention of infection through the corticalis of the petrosa should consist of preparations from cases of otogenic complications; such records are most convincing when they are detected only at postmortem, because in life the ear phenomena had been obscured by some grave syndrome of general disease. In such cases we have before us intact conditions, not altered by any surgical operation or any other kind of treatment. Other suitable cases are operated ones, where the patient has been dismissed as healed, e.g., after antrotomy, but perished later on account of a complication, before any new operative intervention could occur. Finally, fatalities after surgical operations may also be considered; in such cases, however, it is only with great precaution that any conclusions may be drawn. This oft-discussed subject is treated here because of observations made upon various series of sections from cases of the kinds mentioned above.

These sections were examined in order to find the point of entry of the infection, the perforation. Once this was found, it was possible, by going backwards step by step, to reconstruct the whole process which led to the fatality. In this investigation one frequently arrives at a remarkable, paradoxical result: the more thoroughly the investigation is carried out, the more difficult it is to ascertain where the decisive step has occurred. One sees a number of communications between the soft tissues on both sides of the tegmen and finds himself at a loss to decide over which path the deadly infection made its progress. For instance, it may, in the case of a panotitis, be impossible to tell why convenient ways of passage, like the inner auditory canal or aquaeductus cochleæ have remained unused, whereas an insignificant vascular canal has led the infection through the tegmen into the interior of the cranium.

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Hagens (Laryngoscope, 47:768, 1937), pointed out in his report on otogenous meningitis that "the microscopic connecting channels may or may not have transmitted the infection." He emphasized that "in most of these connections definite evidence of infection spreading from the petrosa to the meningeal surface was lacking."

In the case of acute as well as of chronic suppurations, many places may exist where such communication takes place, while at the same time no signs of meningitic irritation present themselves. The most striking examples of such a condition have been furnished by specific inflammatory processes. Thus there were among our patients two who had extended caries of the convexity of the skull, and also in the immediate vicinity of the cochlea described in detail: (Zeitschrift f. Laryngol., 14:280, 1926). The anamnesis and the results of the functional examination showed that the internal ear was seriously affected, such affection, notably, dating back to four and sixteen years, respectively. Nevertheless, the disease which, moreover, actually constitutes an osseous process, did not lead to any involvement of the dura. On the other hand, we may use for comparison preparations deriving from congenital syphilis (described in detail: Oto-Rhino-Laryngologica Japonica, 2:1005, 1929). In these preparations innumerable passages from the diseased bone to the dura can be detected. It is true that the latter is thickened; so it was in all probability also in the two cases with chronic caries which, however, did not lead *ad exitum* and thus did not come up for any histological investigation. The thickening represents a condition of defense, which is evidently successful.

The conditions to which we would call attention here relate to such points of entry through the tegmen as can be demonstrated histologically, but which have again become healed or were on their way to become healed. They present a picture of reversible processes.

The cases utilized in the study of this question are shown in Table I. It is not necessary to discuss them here in detail, as they mostly represent material regarding which publications have already been made; they are enumerated in the table and can be consulted for details. These cases have newly been examined from the point of view of the question under discussion.

Fig. 1 represents the normal conditions. The vascular canal entering from the dura proceeds in the typical oblique direction of all foramina nutritia. One should not forget that this oblique direction is characteristic of all bone channels which lead nutritive vessels from the periosteum into the bone. Here the dura also should be

TABLE 1.

No.	No. of Case History	Age Years	Sex	General Disease	Ear	Micro-organism	Operation	Death After Operation Days	Publication in Detail
1.	406 (1926)	5 mths.	Male	Atrophia. Meningitis diffusa sero-purulenta	Otit. med. ac. suppl. l.s.	?			
2.	Sk.I. (1923)	27	Male	Noma of left half of head	Otit. med. ac. supp. l.s.	Symbios. fuso-spirill.			Acta Oto-Laryngol., 9:344, 1926
3	R.M. (1925)	5	Female	Noma of right half of head	Otit. med. ac. supp. l.d.	Symbios. fuso-spirill.			
4.	S.P. (1924)	38	Female	Endometritis sept. p. abort. Parotit. secund.	Otit. med. et. intern. ac. supp. l.d.	?			Passow-Schaefers Beitr., 25:127, 1927 (Case No. 4)
5.	2557 (1934)	36	Female	Amput. supravag. uteri. Parotit. secund.	Otit. med. ac. supp. l.d.	Bac. pyocyaneus			
6.	591 (1937)	37	Female	.....	Otit. med. chr. supp., cholest. thromb. sin. sigm. l.s.	Streptoc. haemol. Bac. coli.	Op. rad. et. sin. sigm., lig. jugul.	4	
7.	289 (1935)	23	Male	.....	Otit. med. chr. supp., cholest. absc. cerebelli l.s.	Bac. Morgan	Op. rad., op. absc. cerebelli	2½	Orvosi Hetilap, 79: 1249, 1925
8.	1132 (1936)	52	Female	.....	Petrositis, absc. extradur. l.d.	Staph. aur. haemol.	Antrotomia	23	Jour. of Laryng. and Otol., 53:113, 1938

Fig. 1. Normal conditions. Oblique direction of the vascular canal from the dura into the bone. Medullary space filled with loose tissue.

considered as representing a periosteum. The superficial medullary space in the tegmen is open, it shows a thick covering of periosteum, which should be considered as representing an extension of the dura into this space.

Places such as are seen in this figure facilitate the propagation of pathologic processes between the interior of the petrous bone and the dura. The direction of such passage is by no means evident in advance, i.e., one is apt to forget that infections may also progress from the dura towards the interior of the petrosa. This is indeed the case, notably in the channels of greater width. Thus we saw (Aquaeductus cochleæ, als Überleitungsweg eiteriger Infektion, Festschrift for Ino Kubo, 113, 1934) how in the case of a boy of twelve years suffering from endocarditis and pleuritis, who died on the fourth day after a sinus operation, necrotic-purulent masses starting from the diseased dura, penetrated into the cavity of the aquaeductus cochleæ and almost reached the cochlear opening of the latter.

Similar points of ready passage are, however, represented not only by medullary spaces, but in many cases also by pneumatic cells,

Fig. 2. Pneumatic tegmen cell. Thickened mucous membrane as continuation of the dura. Purulent contents. Beginning necrosis of the bone at the edge of the orifice.

as appears from Fig. 2 (taken from Case 4). Here the dura is enormously thickened and extends in this condition into the cell, the wall of which is covered by an extension of the dura. In the open lumen of the cell purulent masses are visible. The opening towards the extradural space is clogged by the thickened periosteum itself, but it is clearly visible how on one edge the bone is already giving way, i.e., beginning to become dissolved. It is accordingly necessary to take into account the fact that shortly this opening will become widened and thus it will be more easy of access.

A third possibility of propagation is represented by the connection between medullary space and vascular canal (Fig. 3, taken from Case 2). Here there is visible in the depth of the tabula of the tegmen a medullary space completely filled with pus. From this space a vascular canal leads out from the tegmen, in the typical oblique direction, below the dura. The path over which the infectious masses have to travel here is long and narrow. It is evident that here the open canal would have offered an easy passage between the soft parts on both sides of the bone.

Fig. 3. Vascular canal running in an oblique direction, forming connection between a medullary space in the tegmen, filled with pus, and the dura.

Fig. 4 (taken from Case 7) permits the closer examination of a vessel in which propagation is taking place. It is thrombosed and the pathologic contents are extending into the dura. Evidences of vigorous repair are, however, already present; the work is going on of closing the dangerous point of entrance. All over the wall of the vascular canal rows of osteoblasts are at work; it is remarkable that it is exactly at the opening at the bony edge that they are most dense. If we imagine this activity as being continued with a similar intensity, one will be led to suppose that a perfect occlusion of this space would have come about within a relatively short time. The layers deposited by osteoblasts are already visible as substantial lamina, they are attaching themselves to the more obtuse angle of the bone opening and consecutively down to the bottom of the portion of the canal through which the microtome went. At the same time the periosteum has become thickened so as to present bulges, which also serve the purpose of occlusion.

Progressing further in our series, we meet processes of repair presenting the appearance of Haversian lamellæ, as for instance, in Fig. 5 (taken from Case 7). Close inspection will enable one to

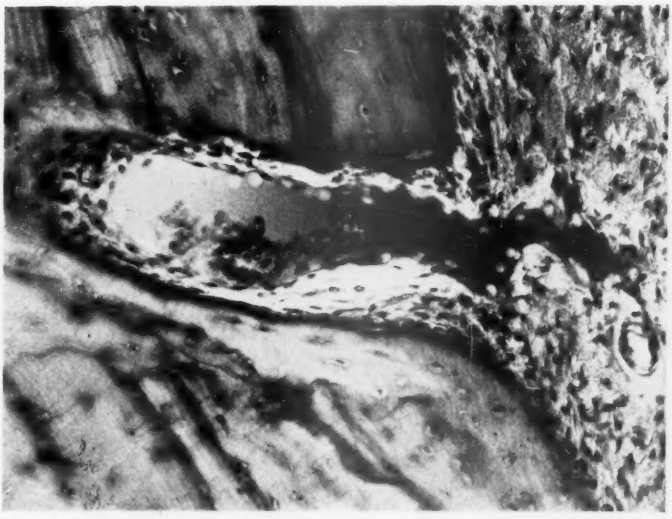

Fig. 4. Thrombosed vessel entering from the dura. Between the vessel and the bone, row of osteoblasts, representing signs of repair.

Fig. 5. Filling block, interrupting, in the form of Haversian lamellae, the covering lamellæ of the tegmen. Hyperemia in the dura.

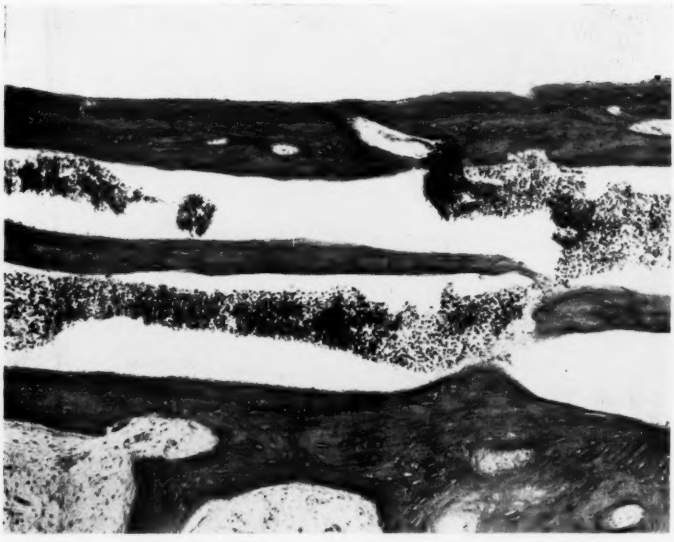

Fig. 6. Path of the progressive suppuration from the deeper into the more superficial layer of cells, finally into the oblique vascular canal towards the dura.

conclude from the high degree of distension of the vessels in the dura and in the bone and from the abrupt interruption of the lamellæ of the outermost covering layer, that here a bony block, a kind of grafting had been produced which may be considered to represent a sign of an endeavor of delimiting the process.

How the progress of the infection takes place from cell to cell in the interior of the tegmen is shown in Fig 6 (taken from Case 1). The tegmen is well pneumatized and shows various rows of cells arranged in layers one above the other. The purulent contents are passing step by step from the deeper to the more superficial rows. It is in the covering of the latter that the oblique vascular canal is situated; it is just about to be passed by the masses of pus. This is a clear example of the passage from pneumatic spaces through a vascular canal. Compare Fig. 3, in which the vascular canal conducted the contents from a medullary space.

Undoubtedly any close adhesion of the dura to the underlying bone will favor the formation of such passages. On the other hand,

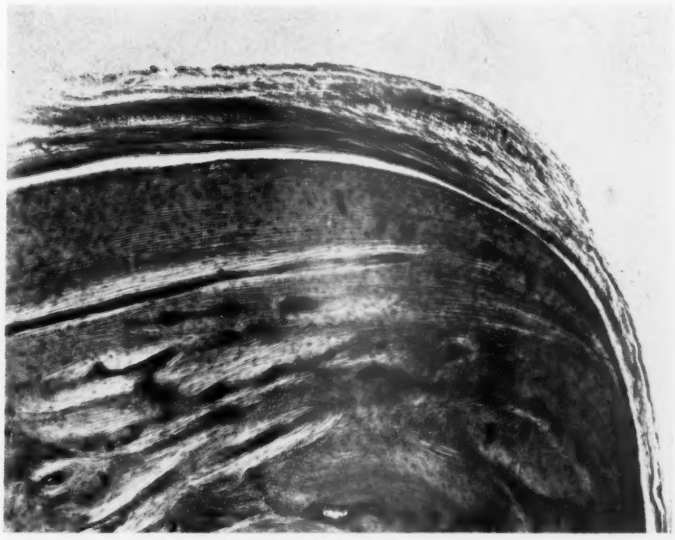

Fig. 7. Strictly parallel position of the covering lamellae of the tegmen and of the fibrous layers in the dura. (Entrance to meatus acusticus internus.)

places where the strata of the superficial bone lamellæ and those of the dura are running parallel with each other should be differently regarded. Here the connection between the two is a less intimate one. Such a place occurs regularly at the entrance into the inner auditory canal, as seen in Fig. 7 (taken from Case 3). In fact, this point is relatively seldom the place of propagation. This may possibly also be ascribed to the circumstances, that it is in the neighborhood of the porus acusticus internus that the most dense, hardest bone of the petrosa is found, as can already be noted during the process of decalcification. This place can be opposed as a "difficult" to those which have in the preceding illustrations been called "easier" places of propagation.

The preceding figures relate to the possibilities of propagation of infection; there follow now three in which it is possible to observe the first stages of the formation of the extradural abscess. Fig. 8 (taken from Case 8) shows a medullary space together with the vessel in which, in addition to thrombus in the lumen of the vessel, the purulent contents penetrate into the deeper layers of the dura.

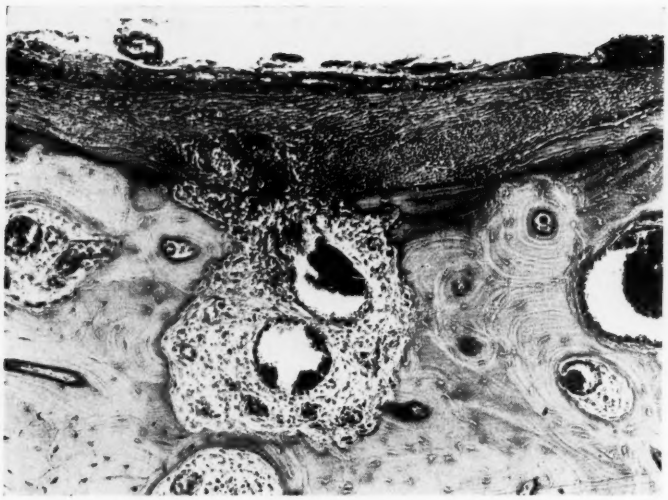

Fig. 8. Medullary space with thrombosed vessel and purulent contents, opening below the dura. Beginning of collection of pus in the thickened dura. Bone attacked at the orifice.

Here it is already possible to notice a thickening, the bone edges also are already attacked, so that the orifice appears to be widened hereby. Above the opening of the medullary space a well circumscribed area of dense infiltration has formed, as a precursor of the formation of the abscess. That repair by closing of the cavity with the aid of osteoblasts is imaginable, is shown by the activity of osteoblasts observable in adjacent medullary spaces.

Fig. 9 (taken from Case 7) shows a far-reaching thrombosis of the dural vessels, whereas the conditions of the propagation from the medullary space are approximately the same as are seen in Fig. 8. We see here small centers of infiltration, all dural layers have become loosened and at the same time thickened. A larger space has already become formed, filled with purulent matter and detritus. Here, also, osteoblasts are visible on the wall of the medullary space, with the aid of which repair can be started.

It is beyond doubt that the conditions as shown in Figs. 2 to 9 are still reparable. This is true of the processes in the soft as well as of those in the bony parts.

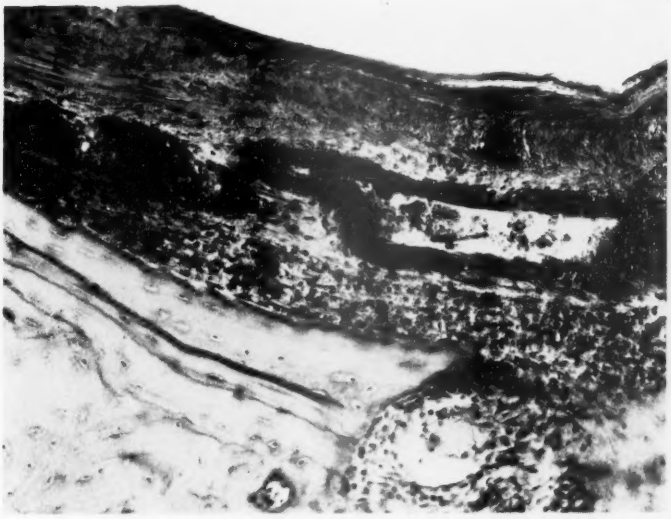

Fig. 9. Medullary space with purulent contents, opening below the dura. Strongly hyperaemic and thickened dura with beginning formation of abscess.

In the soft tissues it is the vessels that play an important part. It is, however, exactly in these vessels that it is possible for a protective occlusion to occur, particularly as the vessels are of small size and become thrombosed rapidly. By recanalization and by the new formation of the whole vessel it is then possible to reach a "status quo ante." How processes of repair are then taking place in the pneumatic spaces after inflammation is, in fact, well known in all their details. We will only remind the reader of the fact that the activity of the mucoperiosteum manifests itself in alterations of the lumen, or, if this expression is preferred, in the alteration of the degree of pneumatization. This means, in the case discussed here, a narrowing, filling up, whereby it is intended to obtain an occlusion from the endocranium. The covering of the soft parts itself also undergoes thickening. Cicatricial tracts are produced, which also contribute to the narrowing of the spaces in question. To these processes in the interior of the petrous bone there corresponds in the endocranium a pachymeningitis; such places betray themselves at the dissection by the fact that here it is more difficult to draw off the dura.

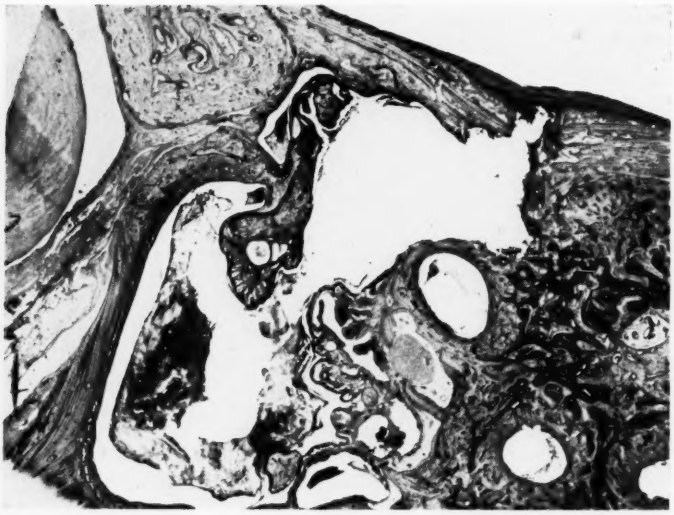

Fig. 10. Necrotic-purulent process in the epitympanum. Rupture of continuity at the bone of the tegmen. Continuity of the dura is still maintained.

The findings represented in the Figs. 2 to 9 represent perforations which have become healed or in connection with which it is not possible to deny the possibility of a spontaneous repair. These are propagations in which the progress of the inflammatory process was inhibited at a certain stage, so that no fatal exit occurred, at least not through the mediation of these places. Accordingly, we would suggest for these processes the name "perforations (propagation) of frustrated form." This expression is intended to cover such cases which undoubtedly represent onsets to a transgression of the corticalis of the petrous bone towards the endocranium, while at the same time still representing reversible, reparable processes.

Finally, three pictures are shown for the purposes of comparison, viz., to show how transgressions are developing in which a reversal is no longer possible and in which a fatal exit can no longer be held up. At the boundary line, so to say, between the two groups stands the dissolution of continuity of the tegmen bone (Fig. 10, taken from Case 7); there the bone has in consequence of the necrotic process become dissolved. The dura still remains unbroken over

Fig. 11. Extradural abscess at the apex of the pyramid, formed by dissolving of osteomyelitic sequestra.

this place also, it is thickened, its vessels are filled excessively. It may be imagined that in the case of a lesser process this relatively small bone defect will again become filled up, particularly if the dura resists and is able to exert its periosteal, osteoplastic function.

The two last examples represent cases of irreparable propagations; nevertheless, they differ from each other in the fact that the same result has been arrived at, in the one case amidst the most intense reaction, and in the other amidst an entirely apathetic behavior of the surroundings. Fig. 11 (taken from Case 8) shows a perforation which has come about in such a manner that the osteomyelitic process has sequestrated the apex of the pyramid and that thus the extradural abscess has become formed. All around, a most intense activity manifests itself in all cavities, even to the stage of sequestration. On the other hand, the perforation which is demonstrated in Fig. 12 (taken from Case 2) has, in the case of a noma, taken place without any reaction in the surroundings; in fact, the absolute absence of any reactive processes is characteristic for this disease, whether soft parts or bones are concerned.

Fig. 12. Rupture without reactive processes in the vicinity: characteristic for noma.

Of course, all these pictures represent momentary conditions which are here inserted into an ideal series. The construction of the latter is, however, based on pathological and clinical experience.

The most important clinical question presenting itself is whether and how it is possible to detect a propagation of this "frustrated" form in the course of the illness.

We have seen that the first result of a propagation of this kind is represented by the beginnings of an accumulation of pus between bone and dura, i.e., by the first histological symptoms of an extradural abscess. The clinical symptoms would accordingly be those which are characteristic for the formation of an abscess of this kind. Now it is a well-known fact that a great number of these abscesses are only detected at operation; thus Watkyn-Thomas and Lowndes-Yates state: "More than one-half of the cases of extradural abscess are unsuspected previous to operation." Kerrison even gives expression to the following opinion: "The usual absence of characteristic symptoms, or our inability to interpret them correctly, is indicated by the fact that the lesion is rarely diagnosticated prior to operation

for the relief of acute mastoiditis." The symptoms: local pain, general headache, and sudden temperature rise, can all be explained by other events. In any case it is necessary to consider these symptoms, in view of their importance, one by one.

As regards the temperature, it is possible that such rise should accompany the first signs of a meningitic excitation through lumbar puncture, seeing that it is participation of the dura that is concerned. In fact, it is possible for the meningeal symptoms to be absolutely predominant (Krepuska). However, as this is particularly the case with extradural abscesses of greater extension, we may disregard these symptoms in the case of the circumscribed minor centers with which we are concerned in the present cases. The same is also true of the slowing down of the pulse, a symptom of pressure which will present itself only after the abscess has reached a certain volume. The place where these breakings through will present themselves with the greatest frequency is the tegmen antri; this is the most usual starting point for extradural accumulations of pus. This is, however, also the place where the probability of a possible spontaneous cure is the greatest, because it is this point which is at the greatest distance from the apex of the pyramid. In fact, if one proceeds further and further towards the interior, one must reckon more and more with the difference which exists between the propagation from a pneumatic cell and that from a medullary space. (For details see: Jour. of Laryng. and Otol., 53:112, 1938.)

Similarly, the symptoms of general headache will mean just as little, because this may be due to many other conditions. More significant will be the localized pain in case it presents itself in the manner usual with the purulent processes of the petrous pyramid. In such cases it will be possible to suspect that the breaking through the tegmen of the inner half of the pyramid is beginning.

Characteristic is the multiplicity of these places; this is the most important feature in the individual case history. This is related to the conditions of pneumatization of the petrosa. Notably, even though pneumatic cells are very frequently found in the apex of the pyramid, nevertheless the degree of pneumatization diminishes in most cases proceeding from the antrum towards the apex. To this topographical factor is added a time factor. If the attempts at propagation are taking place at long intervals, the dura acquires a substantial degree of capacity of resistance. The dura is, so to say, immunized by small doses. If these doses, on the other hand, follow each other in rapid succession, the attack in question has to be considered a multiple one which the meninges will resist with greater

difficulty. As, corresponding to the nature of otitic propagation, the inner parts are usually those to be affected latest, it will, with the progress towards the apex, become more and more likely that the pneumatic propagation will be followed by an osteomyelitic one, because towards the apex more and more medullary spaces take the place of the pneumatic cells. If, then, from the medullary spaces of the apex a propagation dangerous per se will occur, the meninges will give way all the more easily, because they already present a number of weakened points. We should call this factor the pathologico-anatomical one. Thus topographical, time and pathologico-anatomical factors will contribute jointly to the greater importance of the propagations.

As regards the therapeutical conclusions which can be drawn from the facts set forth above, the following should be said: The possibility of a spontaneous repair should under no circumstances induce us to wait longer than usual when the existence of such an endangered point may be suspected; particularly not as regards palliation measures. Most probably such occurrences have already frequently played a part in the evaluation of therapeutical measures in the following sense: the symptoms arising at the beginning of the process of propagation disappear after the spontaneous repair; after a disturbance of suitable length, the general aspect of the illness again assumes a less acute form; this relaxation has, however, certainly frequently been attributed to the therapeutical measures taken, such as, for instance, ligature of the jugular vein, permanent drainage of the spinal fluid, chemo-therapeutical administrations, etc.

At the occasion of the operation it is difficult to convince oneself of the existence of these places, as they are microscopic. In the histological analysis it is possible to see again how the lethal rupture was preceded by several and indeed numerous other onsets, which, however, were arrested by those obstacles discussed above.

The difficulty of the diagnostic and therapeutic evaluation of these data consists in that histological preparations of nonoperated material of intracranial complications is relatively rare. Further, it is in general more difficult to find the corresponding clinical pictures for certain pathological facts than to find convincing pathological evidence for certain clinical processes.

Many interesting facts come to light from the observation of these reparable, reversible propagations of frustrated form. It may be expected that further observations will bring further important information.

SUMMARY

1. In the histological examination of petrous bones after inflammatory diseases which were accompanied by intracranial complications, there are found on the tegmen, between the dura on the one side and the soft parts of pneumatic spaces or of medullary and vessel spaces on the other side, places of passage, where an attempt at perforation took place, but where such rupture was prevented by processes of repair.

2. These microscopic, still reversible, reparable processes which may be called propagations of frustrated form, will present themselves in the course of the disease as exacerbations of the latter. Their symptoms are in many respects identical with those of other complications; accordingly, their disappearance after spontaneous repair is often attributed to therapeutical measures, like ligature of the jugularis, or chemo-therapeutical measures undertaken on the assumption of some other more familiar complication.

3. During the course of the disease a number of propagations of frustrated form may present themselves. If these follow each other at longer intervals, they will impart a certain immunity to the dura, while if they present themselves in rapid succession, they will create a number of weakened places on the dura which may be fatal in the case of a new attack. This is particularly the case if the new onset is an osteomyelitic one, for instance, coming from the apex of the pyramid. In this case the previous defects will, by summation, produce a fulminating infection.

XXXII

OSTEOMYELITIC INVASION OF THE FRONTAL BONE FOLLOWING FRONTAL SINUS DISEASE*

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PHILADELPHIA

We are all familiar with the numerous opinions on this dreaded complication of sinus disease, but we must thank Furstenberg (1931) for clarifying and elaborating on McKenzie's paper of 1913, and in showing us the necessity of wide bone extirpation beyond the visible limitations of the infections, thus preventing retrograde thrombosis, epidural and brain abscesses.

In 1933, Judd and Mosher stressed the line of edema of the scalp as a rough outline of the underlying bone infection. They also proved that demonstrable roentgenographic findings are from seven to ten days behind the infected area. In 1936, Mosher,[1] in confirming his earlier findings, made the statement, "Osteomyelitis writes across the patient's brow not only the diagnosis but the treatment. Give me a swimming case or one due to an open windshield and I am going ahead."

McKenzie divided osteomyelitis into the fulminating or malignant, and the benign or self-limiting types. Mosher states, "I haven't the fortune-telling ability to tell which these are."

The history and physical findings of the case should give us our lead, even though our roentgenograms are negative. I have found that tri-weekly roentgenographic study will generally show a progressive picture of this disease. Suspicious looking areas in the bone will appear gradually on the later films. These alone should lead us to suspect a beginning calvarium infection. If, in an acute or chronic frontal sinus disease, we even suspect that the infection has spread into the bone, we should not hesitate to remove the entire anterior wall of the frontal sinuses from the superciliary ridge to its union with the posterior wall.

Furstenberg,[2] in his recent paper describes osteomyelitis as "the ravaging, devastating, progressive invasion of the osseous tissue—of the dura—and of the brain. In a series of forty-six cases of

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*Read before the College of Physicians of Philadelphia, October 19, 1938.

diffuse osteomyelitis of the cranial bones, in all but eight cases the calvarial infection followed an operation upon one or more of the nasal accessory sinuses. In most instances the Killian fronto-ethmoidal operation preceded the diffuse infection of the skull, although the intranasal procedures were also apparent forerunners of the grave complication. Intranasal drainage of the antrum and the Caldwell-Luc technique have also been followed by devastating invasion of the calvarium." Medical history confirms his findings.

Behrens,[6] in a review of osteomyelitis, states that the exciting cause is usually trauma, either operative or accidental, or an acute exacerbation of a chronic infection with the staphylococcus as the most frequent offender. Most patients are under thirty and that women are affected more frequently than men. Swimming also has been recorded as a common factor.

Just how do our records compare with his in the cases reported?

1. D. M., female, age 13. History of scarlet fever at the age of 10. No history of sinusitis, swimming or injury.

2. W. W., male, age 25. No history of sinus disease or injury. Swimming and driving with open windshield seen to be the etiological factors of his sinus infection. Trephine and drainage of the frontal the cause of his osteomyelitis.

3. M. M., female, age 15. No history of sinus disease but frequent head colds. No operations or accidents. Swimming the factor.

4. R. L., male, age 19. Same history as M. M.—no sinus history.

5. L. M., female, age 10. Trivial blow from brother (age 2) over left superciliary ridge. No sinus history.

6. F. S., female, age 38. History of acute frontal sinus disease; rupture at upper inner angle. Incision with gauze drainage. Three years drainage. Osteomyelitis, brain abscess. This patient was 6 feet 1 inch in height and weighed but 68 pounds. She died three hours after operation.

We believe firmly that the majority of osteomyelitis of the calvarium following sinus surgery is due to inadequate surgery at the original operation.

In a recent conversation with Dr. Mosher, he said, "I am not positive of my figures, but I think 50 per cent of our osteomyelitic cases were diagnosed previous to our sinus surgery."

If we can include osteonecrosis of the frontal bone, we can confirm such a statement.

We know that the thinnest portion of the orbital plate of the frontal bone is around its articulation with the lamina papyracea of

the ethmoidal bone; therefore, one would think that this lowest point of the floor would be the natural site for an abscess to point.

It has been our experience to find the point of rupture in the roof of the orbital plate rather than at the inner angle of the eye. There have been several perforations in the outer third of the sinus floor,[1] others in the mid-third. In a few, the perforation has occurred in the anterior wall, at the upper inner angle of the sinus cavity. I do not recall any fronto-orbital abscess perforation through the lower inner end of the floor. In every orbital abscess at the inner canthus, the probe has found its way into an ethmoidal cell, although the frontal sinus was found to be diseased at operation.

Davis[2] states that 72 per cent of orbital complications are due to rupture of the orbital wall from a suppurative sinusitis. We agree with his findings. The frontal predominates in adults, the ethmoid in children.

A fronto-orbital fistula is actually a rupture occurring through the sinus floor into the orbital cavity, due to a spreading osteonecrosis. It is my opinion that the continuity of tissue and the infecting organisms spreading by contiguity through the Haversian system allows the infection to penetrate through the compact bone to reach the cancellous bone.

In those frontal sinuses in which the walls are of pure compact bone, the first evidence of osteomyelitis is found generally at the junction of the anterior and posterior walls, as there is always cancellous bone with its numerous channels to broadcast the infection from this point. We think that in acute frontal sinus disease, conservatism should be the rule until the temperature and other acute symptoms subside, provided internasal drainage can be established without incision. If the intrasinus pressure is so great that surgical drainage is essential, we do not believe that a small trephine opening for drainage is good surgery, as we have seen too many osteomyelitic extensions following this procedure.

The Jansen operation with its modifications by Lynch, Sewell, Ferris-Smith and others, has been successful in their hands, and our own George B. Wood prefers the removal of the floor to trephining through the anterior plate in acute frontal infections. They believe, and they may be correct, that osteitis is not as apt to occur when the incision is made through compact bone.

We think there is greater danger of bone infection when the cut edges of the trephined openings are in constant contact with the drainage, than there is when sufficient bone is removed to cleanse the

entire sinus cavity. Following this, the remaining bone can be removed in a clean field.[1]

It is impossible to inspect or curette all of the numerous cup-like depressions, and slit-like areas, at the junction of the plates, until the anterior sinus wall has been removed. In numerous instances we have found a beginning osteomyelitis in these areas and it is only by the complete removal of the anterior wall that the "ravaging, devastating, progressive invasion" of this dreaded disease can be checked.

Furstenberg states: "The pathways along which infection may travel are: by continuity of tissue, by eroded diploe, and direct extension of infection to the frontal diploic venous system, producing a septic thrombophlebitis and the typical osseous changes of osteomyelitis between the outer and inner tables of the skull."

Anatomy shows that the frontal diploic vein converges downward and forward to exit through a small channel in the superciliary ridge. This vein exposes the diploic venous system to any suppurative processes arising within the soft tissue following the Jansen or Lynch operation; i. e., stitch abscess around the orbit. This danger of spreading the infection into the brain is one of our objections to the so-called fronto-ethmoidal operations.[1]

The great bulk of the venous drainage of the skull pierces the inner table to empty into the meningeal veins and the blood sinuses, thus any infecting exudate reaching these blood channels can produce a thrombophlebitis of the efferent diploic channels and by retrograde thrombosis infect the bone.

It has been proven at postmortem that a septic thrombus in a vein can cause an exudate over the dura far in advance of any evidence of infection within the bone, thus proving that the changes upon the dura are not simply secondary to the disease of the bone.

Again, from our anatomy, we find the great bulk of the blood supply to the cranial vault comes from the meningeal arteries. They vascularize the dura matter and send a dense network through the internal table to nourish the bone. The external periosteum sends a meager supply of blood to the bone.

It is plain to see how any inflammatory exudate on the dura can by even a simple swelling cut off the blood supply to the calvarium.

Furstenberg[2] says, "The inner plate of the calvarium has been the first to be destroyed. This may be the only part of the bone

primarily affected. The medullary and external plate may be free of infection." To his mind this proves that the primary invasion and spread of infection is between the dura and the inner table of the skull.

Students ask: "Why does an acute frontal sinus infection terminate in osteomyelitis and brain abscess more frequently than all of the other nasal sinuses combined?"

The answer is: The veins *(venæ perforantes)* of the mucous membrane lining this sinus communicate directly with the dural veins. These veins may participate in a septic thrombophlebitis, which in turn may distribute the infection into the bones of the skull, dura or brain.

The malignant type of osteomyelitis seldom occurs in chronic frontal sinus disease of a long duration, the inhibitor being the thickened compact bone lining the sinus cavity. This osteonagenesis is the result of a stimulation from a low-grade infection. If the period is of sufficient length, this progressive osteitis, by its growth of compact bone, can completely obliterate the frontal sinus cavity,[5] thus walling off any infection within this cavity.

When an acute exacerbation of a chronic infection occurs in a sinus whose walls are of increased thickness and density, osteonecrosis may occur in the thickened bone. If our twins, i. e., virulence of infection and resistance of the patient, are brought back to their normal state before the infection can break into new tissue (cancellous bone), the thickened bone prevents the dissemination of the infection. We therefore have the self-limiting or benign type of osteomyelitis that responds so nicely to conservative surgery.

It is the encountering of several cases of this type of the disease that has led many writers to advise simple incision with the removal of the sequestrum and local drainage. On the other hand, if the patient's lowered resistance with an increased virulence of the organism persists long enough to allow the infection to break through the bony barrier and reach the diploe, conservative surgery is futile. We are now dealing with a rapidly spreading infection with progressive septic thrombophlebitis, inflammatory exudate, and collections of pus between the dura and the bone which tend to cut off the blood supply to both proximal and distal portions of the frontal bone.

We believe in playing safe and assuming that all acute frontal sinuses that require operation probably have some extension of the

infection. We expect to find osteomyelitis, and we believe in a thorough curettage of all softened bone and hemorrhagic osteitis.

We do not hesitate to expose the dura by removing the posterior plate of the sinus. We have found that acute frontal sinus infections, especially swimming cases, rapidly develop an acute osteomyelitis. Others apparently subside with an amelioration of symptoms, and remain comparatively symptom-free for weeks, even months, then suddenly develop a swollen upper eyelid or an exophthalmos. The family physician generally orders orbital compresses, and when the pus points, opens what he believes to be an orbital abscess.[3] When the drainage persists, he seeks the advice of an otolaryngologist who must diagnose the case.

Just what has occurred during this interval? Any infecting organism that is capable of penetrating compact bone will finally reach the diploic bone. With invasion of the diploe, we may expect all of the dire results of osteomyelitis, with dural and brain abscesses, and it is only by the most heroic treatment that we can hope to save these patients.[1, 2]

Briefly, to summarize our treatment, we prefer the trans-eyebrow incision; the elevation of the periosteum skin-flap to the limitations of the sinuses; small central trephine for probing; the Killian V chisel for incising the bone above the superciliary ridge; rongeurs for the removal of sufficient bone to curette and cleanse the sinus cavities; antiseptic bath of the sinuses; then, the removal of all of the remaining anterior wall.

This procedure allows a complete detailed inspection of the entire sinus cavity, and if osteomyelitis is seen, it can be handled in accordance with the judgment of the operator. We believe in pre- and postoperative blood transfusions, and we have found bacterial antigen helpful.

The periosteal skin-flap should be closed by through and through sutures. It is a routine to place rubber tube drainage through an enlarged ostium for drainage through the nose. In severe infections, any additional drainage should be instituted at the outer corners.

If osteomyelitis, dural or brain abscess has necessitated the removal of the posterior wall of the sinus, the exposed dura is covered with strips of sheet rubber, which in turn is covered by a single layer of gauze which is kept saturated, every second hour, with the lysated

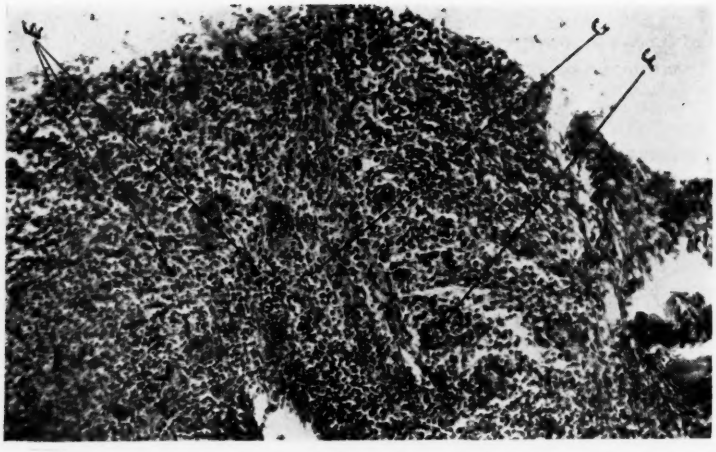

Fig. 1. From the necrotic portion of the bone, showing the inflammatory exudate. The bony trabeculae of the diploë have been destroyed. *E*—Osteoclasts; *F*—Blood vessels; *G*—Inflammatory exudate made up of polymorphs, etc.

organismal fluid. The periosteal skin-flap is closed over the dressing with drainage through the temporal incision.

When the wound is clean at a secondary operation, thin bone specules may be chipped from the edges of the healthy bone and scattered over the dura; these are covered by the external periosteum in the final closure of the wound. We have tried this in one case with remarkable regeneration of bone. The roentgenograms show the result to be satisfactory. The section of the frontal bone was removed as one piece, and thanks to Dr. Eugene Case's careful decalcification and painstaking care, I am able to show you osteomyelitis in five different stages in a single section of the frontal bone (See Figs. 1, 2, 3, 4 and 5).

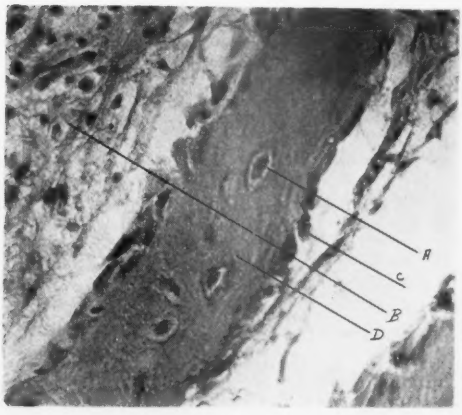

Fig. 2. High power picture of new bony trabeculae. *A*—Bone corpuscles; *B*—Bone marrow; *C*—Osteoblast; *D*—Newly formed bony trabeculae.

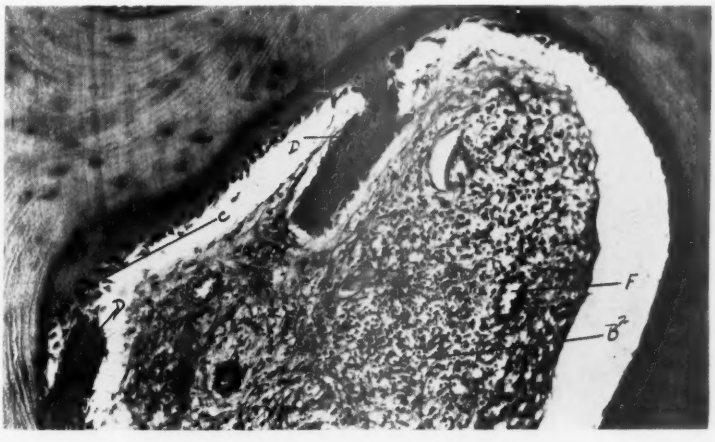

Fig. 3. Newly formed bone in the diplöe, within a marrow cavity near the necrotic (inflamed) portion. B^2—Bone marrow showing the cellular exudate of inflammation; *C*—Osteoblasts; *D*—Newly formed bony trabeculae; *F*—Blood vessels.

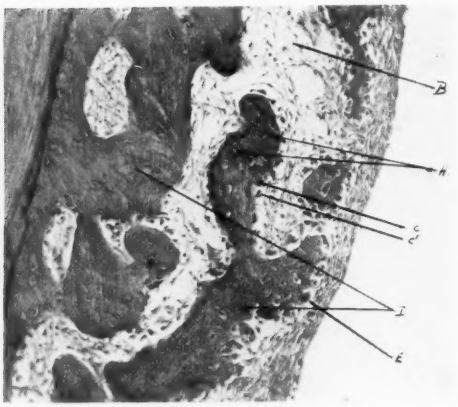

Fig. 4. Newly formed periosteal bone. *A*—Bone corpuscles; B^1—Bone marrow (fibrous); *C*—Osteoblast; C^1—Osteoblast in mitotic division; *D*—Newly formed periosteal bone trabeculae; *E*—Osteoclast.

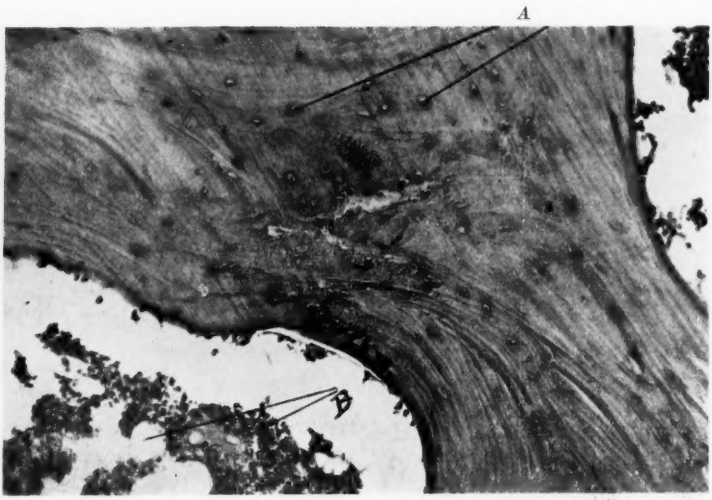

Fig. 5. Bone from the non-necrotic area. *A*—Bone corpuscles; *B*—Bone marrow.

REPORT OF CASES

CASE 1.—W. W., age 25; referred with pansinusitis. History: swimming, then driving with open windshield. In three hours pain and swelling of left upper eyelid. Headache, thick, pinkish postnasal discharge. Palliative treatment for 13 days; some improvement. Acute recurrence on 14th day with increasing symptoms, especially pressure pains over the frontal area.

October 2, 1937: Dr. W. made trephine opening 2 cm. in diameter at the inner angle of left eyebrow. Copious purulent discharge. Gauze drainage. There was a gradual improvement with cessation of drainage.

November 1, 1937: Skin closure of wound.

November 14, 1937: Amputation of Anterior end of left middle turbinate. Irrigation showed purulent maxillary sinusitis.

Present History: Diplopia, vertigo, pain in left ear, neck and shoulder. Transient blindness in both inner fields; parents report periods of mental dullness. This patient's otolaryngologist (a close friend) requested me to operate on the left sphenoid and ethmoid, which was completely hidden by a badly deflected septum.

December 3, 1937: Left spheno-ethmoidectomy: ethmoidal cells badly diseased; sphenoid appeared clean.

December 13, 1937: Left Caldwell-Luc, finding great amount of pathology; right window resection.

December 18, 1937: Wounds healing normally except for brownish, thin pus in left maxillary.

December 20, 1937: Patient reported by family physician confined to bed with influenza; he states sinuses are much better.

December 21, 1937: Patient delirious; temperature, 98; pulse, 72; respiration, 22.

December 27, 1937: Patient led into office apparently blind. Left naries showed free pus and crusts. Operated ethmoid, sphenoid and maxillary areas seem to be healing normally. Pus coming down from left frontal area. Suspect brain abscess, possibly osteomyelitis of frontal bone. Hospitalized.

December 28, 1937: Quoting from Dr. Grant's report: "Spinal fluid 400 water pressure; 18 cell count; 80 per cent polymorphonuclears; blood, 4,600,000 reds, 12,000 whites; temperature, 101 degrees; pulse 68. Seven dioptus choked disk, binasal hemianopsia, left hemiplegia in face, arm and leg. Since Dr. Skillern is sure the abscess is on the left side, a ventricular tap was made. Impossible to reach left ventricle; right easily tapped, about 25 cc. fluid (under pressure) was removed."

December 29, 1937: Trans-eyebrow incision, second mid-vertical incision up to hair line, left frontal bone quite pale. Dr. Grant made small trephine above left frontal sinus to palpate abscess capsule at 2½ cm. in depth of brain in left frontal lobe. Anterior walls of both sinuses removed. Left was soft and necrotic in spots. Sinus cavities filled with pus and loose tissue. Posterior wall of left frontal sinus was a loose sequestrum. Dura covered by granulations. Wound packed with temporary closure of flaps.

December 30, 1937: Abscess tapped by Dr. Grant; "25 to 35 cc. odorless, greenish pus escaped under considerable pressure." Rubber tube drainage of abscess, gauze pack over dura.

January 6, 1938: This case is purely a neurosurgical condition. Dr. Grant's second search of the brain revealed a larger abscess deeper in the frontal lobe, which was drained. This was a hopeless case. There were multiple brain abscesses in formation.

This is the fourth case of osteomyelitis with intracranial complications following the trephining of the frontal bone for drainage of the frontal sinus. I now believe this to be a dangerous procedure.

CASE 2.—M. M., female, age 15. Acute frontal sinusitis twenty-four hours after swimming in a private pool. Right exophthalmia, with boggy white swelling over right frontal sinus.

Operation: Right frontal opened through anterior wall, several spots of softened bone encountered, pus under pressure, loose membrane, fistula in outer third of sinus floor. Sinus cleansed, fistula enlarged, dura clean, remainder of anterior wall removed. Intranasal rubber tube drainage through enlarged ostium. Left frontal opened, membrane swollen and hemorrhagic; all of the anterior wall and the intrasinus septum was removed. Wound closed; uneventful recovery with minimum disfigurement.

There are several other similar cases that I could report, but repetition is tiresome.

CASE 3.—D. M., female, age 13. Referred by Dr. Robert W. Randall, Royersford, Pennsylvania, to Dr. George Coates with the following history: "First saw her October 4, 1933. Greatly swollen upper eyelid, discharging pus. Temperature 103 degrees. Positive signs of meningeal irritation, photophobia, etc. Was in bed three weeks. Systemic symptoms abated, pus discharge increased. Under gas anesthesia, opened the lid more freely for better drainage. Discharge had become green and very foul. Persisted for two weeks then odor stopped, and has drained freely. X-rays show distinct bone disturbances in the frontal sinuses. Her general condition has greatly improved under treatment."

History from hospital records: "Patient states she was well until October, 1933, when, following a severe head cold, she had dull pains over the right eye, with a heavy white discharge from her nose. This was followed by sudden swelling of the right upper eyelid, which progressed to point and rupture at approximately the center of the upper lid. Denied history of former sinus symptoms. Said she occasionally had a slight headache."

Regional examination January 20, 1934. When this child was seen in the examining room, my first impression was that she was a very sick child and poor operative risk for any extensive surgery. She appeared anemic, emaciated, and so weak that she required assistance in walking to the examining chair.

Removing the bandage from her forehead, brownish pus was seen escaping from a fistulous opening about the center of a greatly swollen right upper eyelid. The orbital contents were pushed downward and outward, and the skin over this entire area presented a peculiar edematous, mottled appearance. The forehead

showed a boggy swelling extending from the superciliary ridge upward approximately three centimeters, the skin over the entire forehead being dead white.

Intranasal examination: The entire Schneiderian membrane was hyperemic and swollen, the inferior turbinates inflamed. The middle turbinates hyperplastic, the right crowded between the septum and the lateral wall so tightly that it prevented drainage from the overlying sinuses. There was sufficient room between the left middle turbinate and lateral nasal wall to allow drainage of thick, creamy-white pus. This was seen only after the removal of the pus which filled the left nasal cavity.

Former x-rays revealed massive frontal sinus pathology.

As this rather superficial examination threw the child into a state of shock, she was sent back to bed for rest and supportive treatment, with a clinical diagnosis of chronic suppurative fronto-ethmoidal disease, massive osteomyelitis of the frontal bone, a right frontal lobe abscess and suppurative left maxillary sinusitis. There is a fistulous opening through the outer third of the frontal floor.

Her chart shows, upon admittance to the hospital, her temperature was 99.3 degrees; pulse 100.

Blood Count	4,490,000	Eosinophiles	1%
Hemoglobin	67%	Basophiles	2%
Leucocytes	9,500	Monocytes	4%
Polymorphonuclears	52%	Lymphocytes	41%

The preoperative x-ray report by Dr. Osmond, January 21, 1934:

"Frontals: Left frontal clouded, right is the seat of considerable pathology. It is clouded and the sinus outline is quite indistinct.

Ethmoids: Marked clouding of all of the cells on the left. Considerable clouding of most of the cells on the right. Posterior ethmoids on each side are only slightly, if at all, involved. Orbital extension, strangely enough, appears to be quite clear.

Maxillaries: Marked clouding on left, moderate clouding on the right.

Sphenoids: Negative."

The Ophthalmological Report—Dr. Peter's Service: January 21, 1934. "Negative eye grounds."

Neurological Report—Dr. Weisenberg's Service: January 22, 1934. "Bilateral Kernig. Signs of meningeal involvement with some chronic meningeal changes. The oculomotor involvement is probably peripheral rather than nuclear."

Operation: January 23, 1934. It was with a great deal of apprehension that Dr. Coates and I started operating, as we both considered the child a very poor surgical risk.

Under avertin-novocaine anesthesia (later, warm ether vapor), an incision was carried across the eyebrows. Adhesions made the elevation of the periosteum difficult, especially over the right frontal area. A fistula was encountered one centimeter above the midline of the right superciliary ridge. The exposed frontal bone appeared unduly white, with scattered mottled areas of softened bone. A

small blunt sound was allowed to drop of its own weight through the fistula for 3½ centimeters into the brain. Slight bleeding but no increase in the flow of pus.

With the Killian V chisel, the bone was incised one centimeter above the superciliary ridge, exposing a bluish tinged membrane filling the sinus. This, at first, was thought to be the sinus mucosa. Great care was exercised when enlarging the bony opening not to injure this membrane. It was this careful removal of the anterior wall of the frontal sinus that proved the salvation of the operation, as this supposed sinus membrane proved to be the dura. Following the removal of the anterior wall, it was noted that the posterior sinus wall had disintegrated completely except for jagged edges along the inner and upper extremities of the sinus cavity. This melting away of the posterior wall allowed the dura to come into direct apposition with the anterior wall of the right frontal sinus.

The left frontal sinus walls were intact. The sinus cavity was filled with necrotic loosened membrane and pus, and cultures proved the infection to be a pure hemolytic staphylococcus albus. Culture from the brain abscess cavity was also reported as hemolytic staphylococcus albus.

Dr. Coates and I, working as rapidly as possible in different areas, removed the skull upward and outward beyond the limits of the edematous swelling over the forehead. A hollow sound then was inserted to rupture the capsule of the brain abscess. There was a gush of approximately 10 to 15 cc. of foul-smelling, brownish pus.

While Dr. Coates inserted rubber drainage tubing into the abscess cavity, I made a trough across the entire breadth of the skull approximately one centimeter below the coronary suture line. This trough was carried through the inner table of the skull to break the continuity of tissue, to prevent the further extension of the infection through the diploe. The child's condition had been growing more and more precarious with the passing of each minute. With a continual warning from the anesthetist that the patient's pulse was growing weaker and weaker, the blood pressure dropping, we hurried the toilet of the wound as much as possible. The anesthetist finally reported the pulse was no longer perceptible, and the breath sounds were very shallow. Strychnia and atropin had been given during the latter stages of the operation. An ampule of coramine and other supportive treatment kept the patient alive until a blood transfusion of 250 cc. of citrated blood and 100 cc. of normal saline could be given, then with temporary dressings the patient was returned to her room more dead than alive. At 11:00 p. m. there was a decided improvement in the patient's condition.

Daily dressings, with the following notes of interest:

January 24, 1934: Outer dressings removed down to the iodoform gauze covering the dura. Set of four small Dakin tubes placed in new dressings for continual bathing of the wound with Dakin's solution.

January 26, 1934: Upon removal of the dressings, the dura was found covered by healthy granulations. Removal of the drainage tube from the abscess cavity allowed the escape of one dram of white, creamy pus. The tube was cleansed and reinserted. The left nose was cleansed by suction, as pus was welling up into the frontal wound from the ethmoidal cells. The area of bone between the wound and trough had the dead white appearance of a skull.

January 30, 1934: There was a decided decrease in the intradural pressure. Very little pus in the abscess cavity or on the dressings. When the separate pack

was removed from the left frontal, pus again welled up from the ethmoid to fill the cavity.

February 2, 1934: The dressings over the brain abscess were comparatively dry. The tube was removed, cleansed and reinserted, even though the abscess cavity appeared to be free of pus. When the packing was removed from the left frontal sinus, the cavity immediately filled with pus, which boiled up from the left ethmoidal area, with an odor of colon bacillus. The partially isolated bridge of bone below the transverse trough was definitely dead. Fresh dressings with Dakin tubes applied.

February 6, 1934: The greatly improved physical condition of the patient warranted the completion of the operation.

Anesthesia—avertin-ether.

Assistants—Drs. Wm. Whelan, Furlong and Herr.

Dr. Whelan prepared the wound by painting tincture of metaphen. The scalp was shaved, followed by a second application of the tincture of metaphen.

The original vertical incision was enlarged upward for five centimeters. With the flaps held back, the periosteum was separated from this central incision down to the temporal areas. The transverse trough was enlarged by cutting away the lower edge of bone, then, by making two horizontal incisions from the trough to the opening of the original operation, this bridge of bone was separated of all bony connection to the skull. I felt that even though a downward dissection would be more difficult, it would be best to go through this clean area for the separation of the dura and longitudinal sinus from this bony sequestrum. The separation was made even more difficult by the numerous adhesions between the dura and skull, especially around the longitudinal sinus, but by painstaking work, this bridge of bone was removed intact without injury to the underlying structures. The upper edge of the bone was incised until free bleeding was obtained. The newly exposed dura appeared to be in a healthy state. The formerly exposed dura was covered by healthy granulating tissue. Protecting this area with one to five thousand bichloride dressings, the anterior ethmoidal cells were attacked through the left frontal ostium, and as many as could be reached were removed by the Grunwald forceps. The ethmoid then was attacked intranasally until sufficient room was obtained for the insertion of a large rubber drainage tube. Both maxillary sinuses were opened by biting away the lateral wall beneath the inferior turbinates. After cleansing the maxillaries and ethmoidal areas intranasally by suction, and inserting a large slotted drainage tube into the left frontal ostium, to facilitate the ethmoidal drainage downward, dressings were applied to the frontal area. A small rubber drainage tube was inserted into the shallow brain abscess cavity, the entire wound covered by iodoform gauze, and after freeing the adhesions, the flaps were brought into apposition along the vertical incision. There had been considerable shrinkage of the skin and underlying tissue, consequently when the flaps were brought down, there was a 2½ centimeter deficiency above the superciliary ridge. Silkworm stay sutures were used to prevent further contraction. Patient was returned to the ward in far better condition than at the former operation, the blood pressure being 130, pulse of good volume, rate 120. Time of operation, 75 minutes.

February 9, 1934: Although the external dressings appeared to be clean, the wound was found covered by a thick, creamy pus when they were removed. There was a fluctuating mass approximately five centimeters in diameter under

the right skin flap, another three centimeters in diameter under the left. Reopening of the skin flap revealed walled-off abscess cavities under each. This entire area was painted with tincture of iodine, and the flaps closed by adhesive tape. The abscess cavity in the brain was apparently empty. The left frontal sinus contained a small amount of plastic exudate. A drainage tube was reinserted into the brain abscess cavity, then the entire exposed dura and the frontal sinus cavity was packed with iodoform gauze. The drainage tube was removed from the left side of the nose.

February 13, 1934: The wound appeared healthier than at any time. Small amount of pus over left supraorbital area. The brain abscess had expelled the tube, which no longer could be inserted.

February 14, 1934: Transfusion of 400 cc. of citrated blood to increase resistance.

February 25, 1934: The wound has been healing nicely. The brain abscess cavity is closed.

February 27, 1934. The wound has remained free of pustular discharge and the abscess cavity closed.

March 2, 1934: There was a plastic exudate covering the entire granulating area. Nose appeared free of all discharge.

March 9, 1934: Plastic closure of wound under nitrous oxide anesthesia.

The skin flaps were dissected loose from the fascia for three centimeters beyond the cut edge of the bone. By a very tedious dissection, the periosteum was loosened from the dura and cut edge of the bone. Contraction had shortened the flaps to one-half of their original size. To gain length, the periosteum was elevated for five centimeters beyond the cut edge of bone, then with the flaps turned upward, the inner surface was scarified by numerous horizontal incisions. Even the stretching of this split tissue failed to cover the dura, leaving a gap of fully two centimeters across the superciliary ridge. Heavy retention sutures of chromic gut kept this membrane under tension, with the hope that further stretching might occur. The skin flaps were two centimeters short, and required scarification of the inner surface. Tension sutures were required to hold skin edges together.

March 16, 1934: Due to the thin skin and tension required to keep the skin edges approximated, several stitches had cut into the skin.

March 20, 1934: Skin sutures were being absorbed, wound healing nicely.

March 23, 1934: Wound entirely healed, with broad scar across forehead. The left eyebrow was displaced about one centimeter, otherwise good result.

October 26, 1934: The child had grown five inches, with a twenty-six pound increase in weight in spite of the tumor-like infected tonsils.

October 26, 1934: Tonsillectomy.

July 11, 1935: Eighteen months without symptoms of illness. The cranial defect appeared to be somewhat smaller. Health excellent.

October 5, 1935: Twenty-one months without symptoms of illness. For the first time, pulsation was noted in the upper right quadrant of the bony deficit.

There was possibly a very slight swelling or protrusion of these parts, but as the child was free of all symptoms, with a normal pulse and normal reflexes, this swelling may have been more imaginary than real.

As medical history shows recurrence of brain abscesses after lengthy periods of apparent well being, we shall continue to watch this child for any future development.

February 5, 1937: Child has continued in excellent health. Very little evidence of closure of bony deficiency.

September 24, 1938: This girl is now 5 feet 10 inches, weighs about 140 pounds, and is in excellent health. In five years the skull has not regenerated except for a horn-like growth 2 x 1½ cm. extending from the left side. Roentgenograms shortly after operation and those taken October 14, 1938, both show an 8-8 10 cm. deficiency of bone over the frontal area.

April 8, 1939: Health excellent.

Case 4.—R. L., white male, age 19. History: Swimming, followed in forty-eight hours by right fronto-temporal headache; later that day right upper lid began to swell with constant tearing. No history of previous sinus disease, nor of any allergy. Stated health has been excellent to date.

August 21, 1937: Admitted to Coates' service through accident ward complaining of headache, stuffy nose and a swollen, painfully throbbing right eye.

Physical Examination: Essentially negative, except for swelling over right orbit, extending upward to include the ciliary ridge.

Intranasal Examination showed swollen, boggy, wet mucous membrane, especially in right naries. Here the inferior and middle turbinate were in apposition with the nasal septum. After shrinkage, yellowish-white pus was seen coming down from under the middle turbinate. Mild suction brought away about 5 cc., but was so painful that this procedure was stopped. Patient stated his headache felt somewhat better and thought the eye did not feel quite as tense after this treatment.

August 21, 1937: Patient treated palliatively by shrinkage of entire Schneiderian membrane, followed by thirty-minute exposure of nasal area and forehead to infra-red rays. Neosynephrine spray twice daily with prontolyn grains XX every four hours. This treatment was carried on from August 21 until September 10, as the patient was apparently improving daily.

Roentgenopraphic Study:

August 25: "Conclusions—Pansinusitis most marked in the frontal region where fluid levels are present in both frontal sinuses, higher in the right."

August 26: "Bucky films with patient in recumbent position fail to show any evidence of osteomyelitis in frontal area at the present time."

September 2: "Films in erect position show increasing amount of fluid in left frontal sinus. No fluid level in right, probably because sinus is completely filled, as there is uniform density over the entire sinus area."

As this was a private patient of Dr. Coates, I heistated to change the treatment when he left town September 10, but further studies were as follows:

September 10: "Films of the frontal sinuses made with the patient in the erect position show increased amount of fluid in the left frontal sinus. The right side is again rather uniformly dense. The superomedial wall of the right frontal sinus is now seen to be almost entirely destroyed and there is a localized area of bone destruction extending a short distance upward due to spreading osteomyelitis. There is also some haziness and mottling about the superolateral wall of the left frontal sinus which suggests beginning osteomyelitis at this time. Lateral views show increase in the destruction of the anterior sinus plate.

Conclusion: Stereoscopic films of the frontal sinuses show spreading osteomyelitis extending from the roof of the right frontal sinus near the midline. There is probably also beginning osteomyelitis involving the roof of the left frontal sinus. There has been increase in the destruction of the anterior sinus plate."

Laboratory Report:

Erythrocytes	4,700,000	Neutrophiles	72%
Hemoglobin	69%	Lymphocytes	24%
Leucocytes	12,000	Monocytes	2%

Cerebrospinal fluid clear.

Urine negative.

Cultures:—Staphylococcus aureus (hem.).

By this lengthy study we were able to watch the progression of the infection from an acute frontal sinus through osteomyelitis to a brain abscess. At no time during the progress of the infection were there any neurological symptoms; in fact, had it not been for the positive x-ray films and the irregular periods of mild fever we might have assumed that the patient was making an uneventful recovery from his acute infection.

I got in touch with Dr. Coates (in North Carolina), who instructed me to operate, but as Dr. Coates was on his way home and the patient seemed to be holding his own, I held off until Dr. Coates could take over.

Operation: September 14, 1937. Operators—Drs. Coates and Skillern. Assistants—Drs. G. Whelan and Gordon. Anesthesia—Avertin, later warm ether vapors.

The usual transeyebrow incision with elevation of the periosteal skin flap. Superciliary bone incision to the outer limitation of the sinuses. The anterior plate of the left frontal was mottled, and softened areas were encountered in its removal. The sinus cavity was filled with free pus and soft, loose, necrotic membrane.

The anterior wall of the right frontal was apparently normal. Its removal revealed free pus, loose necrotic membrane, and in its upper half, brain tissue. When this was removed, an irregular hole 2 x 2 cm. was seen at the upper inner angle in the posterior wall through which free pus was being expelled. There was brain tissue, and either abscess capsule or dura at the outer edges of this perforation. The necrotic bone was removed to expose an engorged dura. The frontal bone appeared dead white around this opening, gradually shading into a normal color about 2 cm. beyond.

Necessitating greater exposure of frontal bone, a new incision was tried for the future cosmetic result: The outer end of the eyebrow incision was carried

Fig. 6. (Case 4.) Final closure 72 hours after operation. This picture was taken 48 hours later.

backward and upward just in front of the temporal fossa into the hair line, then upward for 4 cm. This gave ample exposure and the turned-back flap remained in position without assistance. While one operator placed drainage tubes in the 3 cm. deep brain abscess, the other started the bone incision 4 cm. beyond the enlarged perforation. One operator working on the right, the other on the left, a bone flap was outlined from the superciliary ridge upward to the midline of the skull. This trough was deepened until the entire thickness of the skull was incised.

Wishing to remove the bone as a single piece, that a microscopic section might be obtained through its entire length, separation was made from above downward through this clean field with fresh instruments.

This procedure made the removal of the bone far more difficult (than piecemeal removal would have been) over the superior longitudinal sinus, due to the deep sulcus sagitallis and the numerous adhesions.

With the obliteration of the frontal sinuses and with rubber drainage tubes in the nasofrontal ducts, the dura was covered with one-inch strips of rubber tissue, over which gauze was laid. The dressings and brain abscess cavity was then sprayed with staphylo-lysate (Lilly). The periosteal skin flap was brought over the packs and the wound closed by interrupted dermal sutures. The abscess tube drainage and the gauze drainage were brought out through the right tem-

poral area. Through this opening, the gauze and tube were kept moist every two hours with the lysated fluid. There was a postoperative drop in temperature to 97.2 degrees, following which the temperature never exceeded 100 degrees. At the end of seventy-two hours, due to lack of drainage and rapid recovery, it was decided to remove the packing and close the wound.

We have a moving picture of this last procedure which also shows the x-ray studies, the postoperative results, and x-ray plates demonstrating the complete regeneration of the frontal plate.

September 17, 1937: When the dressings were removed, the dura and brain abscess were free of pus. The dura of normal color was covered by healthy-looking granulations and the cut edges of the bone appeared normal. We were not overly optimistic of this apparently clean wound, as it was our first experience with the lysated organism, so to be on the safe side, we made a stab wound over the abscess cavity for further drainage. Then we attempted what we believe to be an original thought in skull surgery.

Being familiar with the writings on the regrowth of bone from bone chips or dust, we chipped off very thin slivers of bone from the thickness of the cut edges of the healthy bone. These were scattered over the dura, and with a final bath of the wound with lysate, the soft tissues were completely closed except for the rubber tube drainage through the stab wound into the brain abscess cavity.

With the exception of several stitch abscesses in the eyebrow incision, an uneventful recovery occurred until the sixth day (September 23, 1937), when, with the removal of the dressings, pus was seen around the tube in the stab wound and a small fluctuation swelling was noted to the left of the midline just below the upper cut edge of the skull. Culture: Staphylococcus albus; non hem.

Thinking this might be a second brain abscess working its way to the surface, a watchful waiting policy was followed for three days. The tumefaction having increased with an increase in the pustular discharge, a large asperating needle was inserted into the mass and 15 cc. of heavy, cream-like pus was removed. Believing some heavy opaque substance might demonstrate the source of the pus on the x-ray plate, 10 cc. of lipiodol was slowly injected into the cavity. The plate showed a simple collection of pus directly beneath the skin, the evacuation of which allowed a rapid, complete recovery with a minimum disfigurement. The brain is protected completely by a regrowth of bone filling in the bone deficiency in the skull.

Comparing this with Case 3 operated January 23, 1934, whose recent x-ray films demonstrate the same bone deficiency as seen in the films taken directly after her operations, seems to us to warrant bone chip transplants. When we consider two identical cases, one without lysated organismal medication that required an open wound with continual pustular drainage for three months, in comparison with another treated with the lysate, the wound being free of pus in forty-eight hours, permanently closed in seventy-two hours and discharged in less than thirty days, we feel that this treatment deserves further trial. "One swallow does not make a summer."

Finis Coronat Opus: The moving picture taken September 16, 1938—fourteen months after operation—has been passed by The American College of Surgeons and can be borrowed by the medical profession upon written request to the author.

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XXXIII

THE USE OF RADIUM IN MAINTAINING A PATENT FRONTONASAL OPENING IN EXTERNAL OPERATIONS ON THE FRONTO-ETHMOID GROUP OF SINUSES: A PRELIMINARY REPORT*

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The problem of the patient who has pansinusitis, and especially that of the patient who has marked hyperplasia of bone produced by long-standing disease or of the patient for whom intranasal operation has produced the same result, is the most difficult the rhinologist has to solve. Most patients would prefer to run the risk of an intracranial complication rather than to present the unsightly appearance which results from some operative procedures, yet many rhinologists have so despaired of securing a cure by any but the most radical methods that they frequently have advised complete removal of the external wall of the frontal sinus, together with the entire lining membrane, in order completely to ablate the cavity. Thus, from a review of the literature, it is not difficult to arrive at the conclusion that the frontal sinus constitutes an obstacle that prevents a comparatively easy solution to the problem of pansinusitis. It is true that some surgeons do not have a serious attitude in regard to the problem.

REVIEW OF THE LITERATURE

For instance, Simpson in a recent communication, stated that if the frontonasal duct remained open for a month, no further trouble need be anticipated. Lynch made little reference to the problem of patency of the frontonasal duct, merely advising that rubber tubes be left in place for about a week following operation. Other writers have taken a different attitude toward the problem of patency of the frontonasal duct. Sewall, although he stated that many patients

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may be symptom-free for long periods, even when no communication can be found between the frontal sinus and the nose, was nevertheless confident that the good result of the operation depended largely on maintenance of the opening, and he offered a modification of the usual technic to effect it. Smith was so impressed with the importance of maintaining a patent frontonasal duct in external operations on the fronto-ethmoid group of sinuses that he was willing to risk the formation of a cholesteatoma in the frontal sinus by placing a tubular skin graft in the frontonasal region to form a permanent opening.

Anderson, in his review of the causes of failure to secure a good result in external operation on the frontal sinus, stated that the principal reason for failure in the Lynch type of operation appeared to be contracture of the frontonasal duct and subsequent retention of the secretion in the sinus. He also pointed out, however, that failure to do a complete operation, particularly in regard to fashioning an adequate intranasal opening, in regard to the abolition of pockets situated in the outer angle of the sinus in large sinuses, and in regard to removing septa in the sinus, was an important cause of failure. Anderson stressed the importance of resorting to the Killian technic when involvement of the anterior wall of the frontal sinuses was present. Lillie and Anderson also pointed out the necessity of bevelling the edges of the bone surrounding the frontal sinus in the Killian operation, so that pockets between the periosteum and the internal wall of the sinus would not form.

OPERATIVE TECHNIC TO ELIMINATE INFECTION

It is obvious, therefore, that in attacking the problem of suppurative involvement of the fronto-ethmoid group of accessory nasal sinuses, the first consideration to secure a cure should be to perform an operation adequate to allow complete elimination of the disease process. After this has been done, it is also obvious that certain failures will occur as the result of contraction of scar tissue about the frontonasal opening, a contraction which produces closure of this opening. The technic used should provide exposure adequate to enable the surgeon to reach all sites of disease. Consequently, the original incision recommended by Jansen, which runs close to the inner canthus and into and along the brow, is the one that seems the most satisfactory to us. The small incision situated just medial to the inner canthus advocated by Sewall seems entirely unsatisfactory in that it does not allow exposure adequate for the surgeon to reach the lateral extensions of the frontal sinus, or when he is doing the Jansen (Lynch) type of operation, to inspect the interior of the sinus for

partitions. Sewall's small incision is particularly inadequate for removal of the internal nasal spine.

In incising the bone during the Jansen type of operation, preservation of the frontal process of the superior maxilla, as originally suggested by Lillie,[6] to prevent falling-in of the soft parts, with consequent closure of the frontonasal opening, is of the greatest importance. The virtual impossibility of securing maintenance of a patent frontonasal opening in a recurrence of frontal sinusitis, when the original operation has included removal of this process without resorting to a Lothrop technic with an opening into the other possibly normal sinus, has been emphasized to us on several occasions.

After the floor of the sinus has been removed completely, with removal of the external angular process of the frontal bone to obliterate lateral pockets, if such a procedure is necessary, the internal nasal spine should be carefully removed with gouge and rongeur. This brings the opening into the frontal sinus over to the septum and establishes the anterior part of the opening anterior to the tip of the middle turbinate process, providing a straight drop down into the nose. If the middle turbinate process is carefully preserved, we feel that it aids in maintaining the patency of the frontonasal opening. We have found that a frequent cause of failure of operative treatment is the presence of an undiscovered cell in the intrasinal septum, and great care should be taken to see that none is present. Meticulous care in removing even normal mucosa seems an entirely unnecessary refinement, as one of us (Williams) pointed out in a paper written in collaboration with H. I. Lillie.[6]

If, as a result of disease of the outer table or if the surgeon suspects that early osteomyelitis is present, a Killian operation seems indicated, we feel that leaving a broad bridge (with careful attention to the bevelling of the edges of the sinus well back into the frontal bone) will not only uncover small projections of the mucosa of the sinus which might otherwise form cysts at a later time, but will also succeed in producing an almost unnoticeable deformity. When both frontal sinuses are involved, much greater certainty of maintaining a permanent opening from the frontal sinus to the nose can be secured by resorting to the technic of Lothrop and removing the intrasinal septum well down into the septum nasi. Following completion of the operation the surgeon should follow the suggestion of Lynch and place rubber tubes in the region of what was the infundibulum. In closing, great care should be taken to approximate the periosteum, because it is felt that this procedure helps in preventing collapse of the soft tissues of the orbit into the frontonasal opening.

It has been found, however, that in spite of the greatest precautions, exuberant granulation tissue will form in the frontonasal opening and will eventually form a band of scar tissue which may completely occlude the opening.

RADIUM THERAPY TO INHIBIT FORMATION OF SCAR TISSUE

Desjardins has pointed out that radiation always has an inhibiting effect, especially on immature cells. It accordingly occurred to one of us (Williams) that the use of radium in the frontonasal opening following operation would tend to prevent the development of granulation tissue and subsequent formation of scar tissue.

After consultation, it was decided that the use of 50 mg. of radium in a brass and silver capsule, enclosed in rubber to absorb the secondary beta radiation from the brass, left in place for three hours, should be sufficient to accomplish the end in view. The screening consists of 2 mm. of rubber, 1 mm. of brass and 0.5 mm. of silver. The dosage is 1 gm., left in position for nine minutes. This technic was accordingly followed in treating sixteen patients who had had, variously, both unilateral and bilateral external operation on the fronto-ethmoid group of sinuses. An attempt was made to evaluate the results one year later by comparing the results with results obtained in the treatment of forty-seven patients having similar conditions, on whom I operated during the same interval of time without the use of radium. For four other patients whose conditions required secondary operation, due to formation of scar tissue in the frontonasal opening, the same dosage of radium was used after the secondary operation had been performed.

END RESULTS, WITH AND WITHOUT RADIUM THERAPY

Of sixteen patients for whom radium was used following the first operation, good results were obtained for fifteen: their nasofrontal openings would admit a size 14 French frontal sound. One patient who underwent a bilateral operation also had bronchiectasis, together with an allergic reaction in the mucosa; this patient's frontonasal opening on one side was narrowed down by scar tissue so that only a small-sized frontal probe could be introduced. No secondary acute exacerbation in the frontal sinus has taken place, however, during the period this patient has been observed.

Radium was introduced into the frontonasal openings of four patients, following a secondary operation, and the openings remained large enough to admit a size 14 French sound in three of them. Despite fairly adequate openings, the fourth patient of this group con-

tinued to experience occasional acute exacerbations of frontal sinusitis which remitted under the application of hot compresses without necessity for external drainage. Of the forty-seven patients for whom radium was not used following primary operation, there were eighteen patients for whom a unilateral operation was done. Of the eighteen patients so treated, three had closure of the nasofrontal duct by scar tissue, with recurrence of the infection. These patients underwent further surgical treatment and radium was used. In one instance, failure to remove the external angular process allowed a pocket of infection to remain at the outer angle of a sinus which extended far laterally. In addition, marked narrowing of the nasofrontal opening had occurred. Correction of this omission and administration of radium resulted in cure of the disease. Another patient was afflicted by a fistula situated near the inner canthus, and the development of this fistula was secondary to closure of the frontonasal duct, but further operation was refused. Fifteen patients underwent bilateral fronto-ethmoidectomy with no attempt being made to perform a Lothrop procedure. Three of these patients required reoperation on one side because of closure of the ducts by scar tissue and another patient required reoperation on both sides for the same reason. Those patients who underwent reoperation subsequently remained well and had adequate frontonasal openings.

In the series of patients for whom radium was used following a primary operation, the Lothrop technic was not used for six who had unilateral disease and for five who had bilateral disease of the frontal sinus. There was one partial failure in the treatment of a patient who had bilateral disease; this failure has been mentioned previously.

Bilateral operation was completed for eleven patients by removing the intrasinal septum and continuing the opening down into the nasal septum as recommended by Lothrop. For two of the eleven patients the Killian technic was used; for three patients having unilateral disease the procedure of Lothrop was also used. Difficulty was not experienced by any patient in maintaining an adequate frontonasal opening after any operation in which the Lothrop technic was used.

It is at once obvious, then, that difficulty in maintaining a patent frontonasal opening usually is encountered only by those patients for whom a Lothrop type of operative procedure has not been used.

In forty-seven operations on the fronto-ethmoid group of sinuses, following which radium was not used, failure due to closure of the frontonasal opening was the result for about 19 per cent. In sixteen operations on the fronto-ethmoid group of sinuses in which

radium was used postoperatively there was a partial failure in the treatment of one patient because of partial closure of the frontonasal opening by scar tissue, an end-result which produces a percentage of failure of 6 per cent.

It is admitted that in this small series of patients for whom radium was used postoperatively to maintain a patent nasofrontal opening, patients are too few for a percentage advantage to have much significance, because one additional failure would double the percentage of failures. However, the apparent success which radium has achieved in treating these patients lends encouragement to a continuance of its trial.

It is of great significance to notice that the Lothrop technic was apparently of more value in preventing closure of the nasofrontal opening than was the use of radium.

It is noteworthy that not a single patient whose frontonasal opening closed experienced symptoms of trouble sooner than four months postoperatively, and that in every instance in which the frontonasal opening closed, recurrences took place within a year or less.

CONCLUSIONS

1. Lack of success in operations on the fronto-ethmoid group of sinuses is frequently caused by closure of the frontonasal opening.

2. The use of 9 gm. minutes of radium in the frontonasal opening postoperatively is suggested as a means of maintaining the patency of the opening.

3. Adequate technic for performing the operation is more important in securing success than is any other factor.

4. The Lothrop technic appears to be more successful in maintaining a patent frontonasal opening than is the use of radium.

5. Closure of the frontonasal opening will inevitably result in recurrence of symptoms.

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XXXIV

THE EIGHTH NERVE IN RELATION TO THIAMIN CHLORIDE AND NICOTINIC ACID: A COMPARATIVE STUDY*

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SAN FRANCISCO

The dual nature of certain vitamin B complex deficiencies is becoming more and more evident. This has been observed particularly in pellagra. Sebrell[1] states that, "While nicotinic acid cures the skin and mucous membrane involvement, the addition of thiamin chloride is necessary to cure the peripheral neuritis."

Shattuck[2] more recently says: "It is well known that symptoms of polyneuritis may be associated with pellagra." Also, "Although the usual distribution of the lesions in pellagra differ from that commonly seen in beri-beri, the changes appear to be similar in kind. Both clinical and pathological evidence points to a close etiological relationship between beri-beri, Korsakoff's syndrome and pellagra."

The present study was based partly on the above statement of Sebrell, but largely on the urinary analyses made available after Sebrell's statement for the output of B_1 and porphyrin.

In a recent paper on "Nicotinic Acid and the Eighth Nerve: A Preliminary Report,"[3] in which is mentioned the study of thirty cases to whom nicotinic acid, nicotinamide, or sodium nicotinate was used in the treatment, the improvement in many cases was very striking. The age incidence varied from eighteen to seventy-eight years. The dosage varied from 33 mg. to 132 mg., t.i.d., depending on the age. The use primarily was based on the fact that Covell (working under the Coleman grant at the Hooper Foundation for Medical Research), studying the histological specimens of the different deficiencies of the B complex, found a greater amount of nerve degeneration in Jukes' filtrate deficient chicks (dermatitis) than was observed in the other factors (B_1, riboflavin, B_6). No black tongue dog has yet been available for study and it has been assumed that inasmuch as the dermatitis chick and the black tongue dog have an

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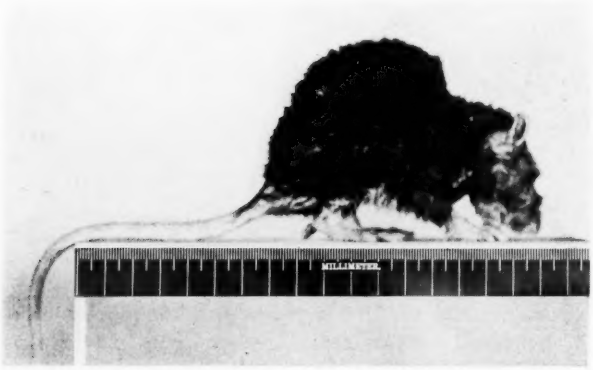

Fig. 1. Double deficiency. Note short thin fur due to riboflavin deficiency and severe lesions on nose, ears and paws, due to vitamin B_6 deficiency.

almost identical pathology, the degenerative changes in the eighth nerve are at least similar if not greater in the nicotinic acid (black tongue dog).

At that time Elvehjem's[4] announcement of nicotinic acid in the treatment of black tongue had not been made and there was only available the crude filtrate factors made from California rice bran which contained some B_6, Jukes' filtrate factor (anti-dermatitis) as well as nicotinic acid.

This material was used in several cases with some evident benefit in the bone conduction curve.[5] Subsequently, when nicotinic acid, nicotinamide, and sodium nicotinate were available, they were used in place of the crude filtrate. These substances were used for several months, with frequent audiograms, and when the hearing curves appeared stationary, thiamin chloride was added and the additional improvement in the hearing will be noted in the case histories to follow.

This same procedure has also been followed in a series of cases of conduction deafness (adhesive and otosclerosis) in young girls and women from the ages of fifteen to forty years, who showed menstrual irregularities and to whom estrogenic substances had been given with definite improvement, not only in the hearing curve, but also in relief of tinnitus (50% of cases) of the roaring type (sea shell), nervousness, menstrual irregularities, etc. In all these cases

B_1 output was low and porphyrin increased in 50 per cent of the cases. In many instances the addition of B_1 and nicotinic acid further improved the hearing curve.

In forty-five cases nicotinic acid, nicotinamide, or sodium nicotinate was used in doses of 66 mg. to 330 mg. daily and continued at such dose.

The very striking fact in these urinary studies is that every case of eighth nerve involvement as well as the obstructive types of deafness were found deficient in vitamin B_1 output. In several cases it was as low as 25 per cent of the normal, and in addition to this, excess of porphyrin was found in fully 50 per cent of the cases. Obviously the method of analysis used is of inestimable value, because it does away with the guesswork method in treatment, and enables one to determine the amounts of B_1 and nicotinic acid necessary to get complete saturation of the body cells. The method of treatment based on the urinary analysis in time may so influence the intestinal mucosa that these substances can be given by mouth until they are absorbed from the food. Unless laboratory studies prove this to be the case, it may be necessary to give these preparations or some preparation containing the entire B complex off and on during the balance of life, especially in cases over the age of sixty or sixty-five years.

The eighth nerve is a peripheral nerve and the cochlea is its end-organ, and is subject to the same degenerative changes as are all the various disease and toxic conditions related to neuritis as listed by Williams and Spies.[6] Underlying nutritional deficiency can explain these nerve changes as mentioned recently by Shattuck,[2] who says: "Moreover, various other neuritides that were formerly attributed to some hypothetic toxin and even certain 'infections,' polyneuritis, are now being attributed to deficient ingestion or utilization of the vitamin B complex." Shattuck also gives a list of the principal causes of polyneuritis similar to that of Williams and Spies.

All the cases observed by myself give a definite history of faulty diets. In the majority of the cases over the age of fifty who show eighth nerve high tone loss in hearing, frequently give other evidence of beginning senescence. Hence the need for the audiometric study and urinary studies by internists of all cases over the age of thirty years applying for physical examinations. Such a study also appears worthwhile in cases showing myocardial degeneration, because many are of the age limits where high tone deafness is found. To further clarify the importance of the vitamin B complex in the human dietary and its need for optimum health, especially its rela-

tion to the nervous system, attention is called to Williams' and Spies'[6] table, "Systems Involved and Lesions Observed in Deficiency Disease in Adults."

I. Skin and other epithelial tissues:

1) atrophy
2) scaling
3) dermatitis
4) pigmentation
5) ulceration
6) cornification

II. Nervous system:

A. Neuritis:
1) pain
2) paresthesia
3) weakness
4) paralysis

B. Degeneration of spinal cord:
1) lateral column
2) posterior column

C. Cerebral:
1) mental disturbances

D. Disturbance of function:
1) tetanty

III. Alimentary tract:

1) anorexia
2) stomatitis
3) glossitis
4) atrophy of tongue
5) achlorhydria
6) loss of specific ferments
7) diarrhea
8) loss of tone of gastro-intestinal tract
9) ulceration of intestine

IV. Hematopoietic system:

1) macrocytic anemia
2) hypochromic anemia
3) microcytic anemia

V. Cardiovascular system:

1) hemorrhage
2) easy bruising
3) edema
4) nutritional heart disease

The question arises frequently: what signs and symptoms are in mild deficiency states? Minot[7] answers the question as follows: "A sense of fatigue or lack of energy, inefficiency, and mental irritability are common. Mild anemia and simple disorders of the digestive tract are the rule. Symptoms and signs of deficiency may be referable to essentially any tissue of the body, but disorders referable to the bones, faulty growth, the gastro-intestinal, neural, blood forming and reproductive system, and the skin are particularly common.

The history may indicate that the patient is nervous, has a poor appetite, mild digestive symptoms, and is fussy and finicky regarding his choice of food * * *."

Another author, speaking of the prodromal symptoms of pellagra, suggests that when present such symptoms call for nicotinic acid. His list is as follows: "Insomnia, loss of appetite, loss of weight, loss of strength, indigestion, diarrhea, abdominal pain, burning sensations from various parts of the body, vertigo, headache, numbness, nervousness, palpation, mental confusion and forgetfulness. There is an erythematous eruption that resembles sunburn with itching and severe burning. It is found on the hands, wrists, elbows, face, neck, under breasts, knees, feet and perineal regions. Later swollen and redder and possibly with vesicles. Later still, swelling decreases and desquamation follows. Tongue and elsewhere reddened, ulcerated, etc."

Recently Peters[8] says: "The antiberi-beri vitamin B_1 was an essential component of many varieties of living cells and was probably universally needed. It is a pyramidon thiazole compound and the most important of the seven B components."

In a group of cases the nicotinic acid in doses of 200 mg. daily was first used empirically before the porphyrin test of the urine was available, and its use was based on the histopathological findings of Covell and Noble in their study of the eighth nerve in Jukes' dermatitis chicks.

In these chicks the physical changes were more rapid than the changes in B_1 acute deficient rats and the nerve degeneration was also more pronounced. No black tongue dogs were available, and it was assumed that the nerve changes could not be less than Jukes' chicks. In the majority of cases nicotinic acid was continued over a period of several weeks, and when the audiometric curve appeared stationary, thiamin chloride was added in doses varying from 3 mg. per cc. to 10 mg. per cc., six days per week, often for several weeks, depending on the age of the patient and his physical response.

This dosage of thiamin chloride is now controlled by urinary tests for B_1. Spies and his co-workers have developed a colorimetric method for determination of nicotinic acid, using the Sheard-Sanford photolometer. Further experimental study is necessary before this or any other colorimetric methods can be accepted as fool-proof. The following methods for determination of B_1 and porphyrin output in the urine have been used in our studies.

Thiamin chloride: The method used in the determination of the twenty-four hour urinary thiamin output is a compilation worked out by R. K. Main, Ph.D., of the Harriman Research Laboratories, Southern Pacific General Hospital, and the analyses herewith reported were made by Dr. Main. The method used was a modification of the thiochrome method of Jansen and collaborators[9] using ferricyanide as the oxidant.[10] After oxidation of the thiamin to thiochrome in alkaline solution it is extracted with isobutyl alcohol. The method as applied to urine by Westenbrink and Goudsmit[11] was used in a few determinations at the beginning of the series. The method found most satisfactory and used in all determinations of thiamin as reported in this paper, with the exception of the few mentioned above, was essentially that of Ritsert.[12] Two five cubic centimeter aliquots of the twenty-four hour urine specimen were made alkaline with ten cubic centimeter portions of 15 per cent NaOH. One, the "unknown," was treated with five cubic centimeters of 1.0 per cent potassium ferricyanide solution, while the other, the "control," was diluted with an equivalent amount of distilled water. Both were shaken for two minutes. Each was extracted by continuous shaking for three minutes with ten cubic centimeter portions of isobutyl alcohol. After sufficient standing, each alcohol layer was pipetted off and filtered. The fluorescence of two cubic centimeter portions of the filtered alcohol layers, when illuminated at right angles to the line of vision by a beam of ultra-violet light, were compared. The control served to estimate the residual fluorescence due to pre-formed fluorescent substances in the urine. Portions, measured from a semi-micro burette, of a standard solution of thiochrome in isobutyl alcohol, were added to the control until the fluorescence of the control and the unknown exactly matched. The amount of this standard solution added is a measure of the amount of thiamin originally in the urine. This standard solution was prepared from a standard aqueous solution of thiamin by an exactly similar manner to that used on the urine. The twenty-four hour urine specimens were kept acid by the presence of five cubic centimeters of glacial acetic acid placed in the collecting bottles. They were kept on ice until the determinations were made.

Porphyrin: The following method used in the determination of porphyrin in urine has been prepared by Jesse L. Carr of the Pathology Department, University of California Medical School, and the porphyrin analyses herewith reported were carried on in Carr's private laboratory. To 25 cubic centimeters of urine add 0.5 cubic centimeters of acetic acid and 25 cubic centimeters of ether. Extract for twenty-four hours in a separatory funnel. To the ether fraction,

add 25 cubic centimeters of 25 per cent hydrochloric acid which extracts the porphyrin from the ether fraction. This second extraction contains other chloride materials as well as porphyrin, which can be removed with two to three washings of distilled water. The fluid is then placed in front of an ultra-violet light source filtered with a Woods filter. A pink fluorescence in the hydrochloric acid fraction is indicative of porphyrin, which is then identified in the spectroscope, the bands appearing about 450 and 500. In the urine and in the ether partition a blue fluorescence will be noted which is due to vitamin B and other substances as yet unidentified.[13]

REPORT OF CASES

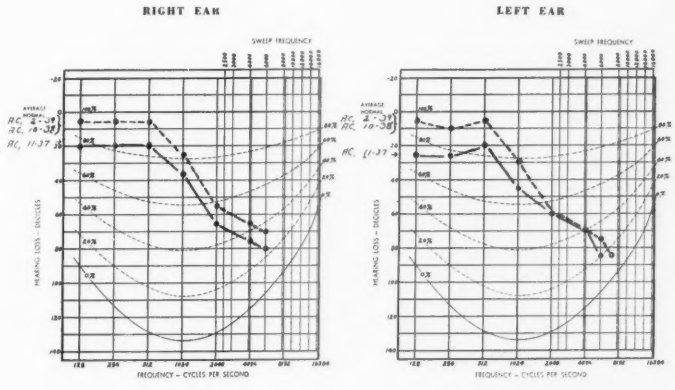

Fig. 2.

Case 1.—Mr. H. E., age 39. Treatment: November 2, 1937, to January 4, 1938, twenty injections of riboflavin; no improvement in hearing following the use of this material. January 4, 1938 to October 24, 1938, nicotinic acid, 132 mg. daily; October 24, 1938 to December 7, 1938, sodium nicotinate, 200 mg. daily. Hearing curve following the use of these materials showed a definite improvement in both ears. December 7, 1938 to February 21, 1939, thiamin chloride injections, 3000 I.U. per cc., one cc. daily. There was no additional improvement in the hearing curve from the use of the thiamin. Both riboflavin and especially thiamin chloride seemed to improve his mental activity, general feeling of well-being and sex activity.

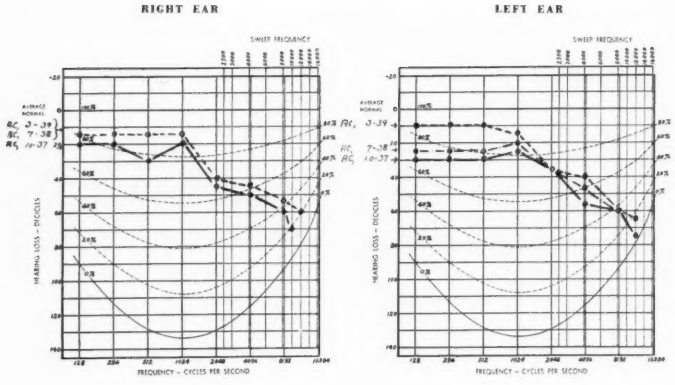

Fig. 3.

Case 2.—Mrs. H. B., age 43. The outstanding conditions in this patient were her anemia (Hgb. 61%) and her deafness of five years duration. Treatment: Use of reduced iron and Lederle's liver extract from October 25, 1937 to May 10, 1938, improved the anemia. May 10, 1938 to August 25, 1938, nicotinic acid, 66 mg. t.i.d. Hearing curve following the use of this material showed improvement in both ears. August 25, 1938 to November 18, 1938, twenty-five injections of thiamin chloride, 3000 I.U. per cc. and nicotinic acid capsules, same dosage as given previously. November 18, 1938 to March 13, 1939, thiamin chloride, 1000 I.U., and nicotinic acid, 66 mg. t.i.d., by mouth. In spite of this dosage of thiamin chloride her urine output on January 18, 1939, was only 17.3 I.U. per 24 hours (normal 20 to 35 I.U. per 24 hours). The only available explanation as to why the B_1 output failed to reach the normal level is that her body cells probably were thoroughly depleted of B_1. On March 13, 1939, there was an additional hearing improvement in the left ear.

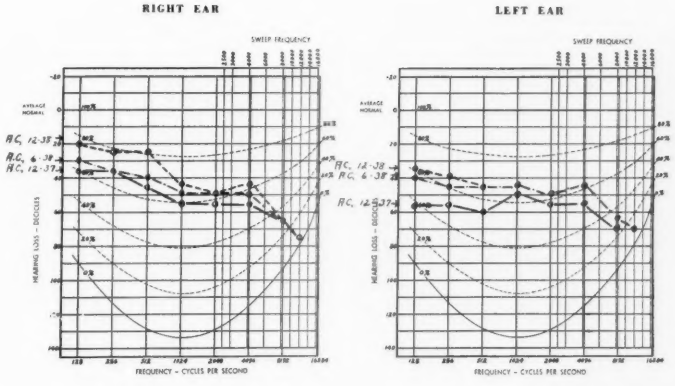

Fig. 4.

CASE 3.—Mr. A. J. H., age 59. This patient has been subject to noise for twenty years, having traveled on railroads nearly 40,000 miles yearly. It is, therefore, not surprising that his hearing for voice has been markedly impaired and that he presents an atypical nerve hearing curve. Treatment: March 3, 1938 to April 26, 1938, twenty-five injections nicotinamide, 30 mg. per cc.; April 26, 1938 to November 7, 1938, nicotinic acid, 132 mg. daily by mouth. Hearing curve following the use of these materials showed a definite improvement in the left ear and slight improvement in the right ear. On November 7, 1938, the dosage of the nicotinic acid capsules was increased to 264 mg. daily and there were added thiamin chloride injections, 3000 I.U. per cc., one cc. six days a week, until February 10, 1939. An audiogram made at this time shows that the gain from thiamin was greater in the right ear and that nicotinic acid produced the greater gain in the left ear. The reason for this reversal of action is not possible of explanation at this time.

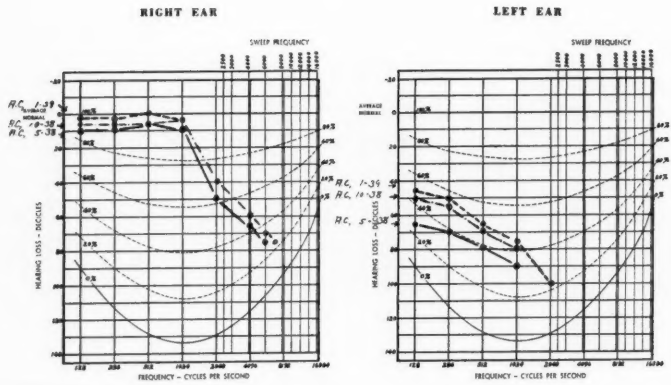

Fig. 5.

Case 4.—Mr. E. V. R., age 60. Treatment: May 24, 1938 to October 24, 1938, nicotinic acid, 66 mg. t.i.d., by mouth. Hearing curve at the end of this time showed three notes of the right ear had improved five decibels and four notes of the left ear had improved twenty decibels. On October 24, 1938, thiamin chloride injections, 1000 I.U. per cc., one cc. six days a week were added and continued until January 25, 1939, at which time the audiogram showed three more notes of the right ear had improved five decibels and note 2048 was now audible by the left ear. Here it also seems that the increase in hearing is definitely due to nicotinic acid plus thiamin chloride, and illustrates the dual nature of the deficiency.

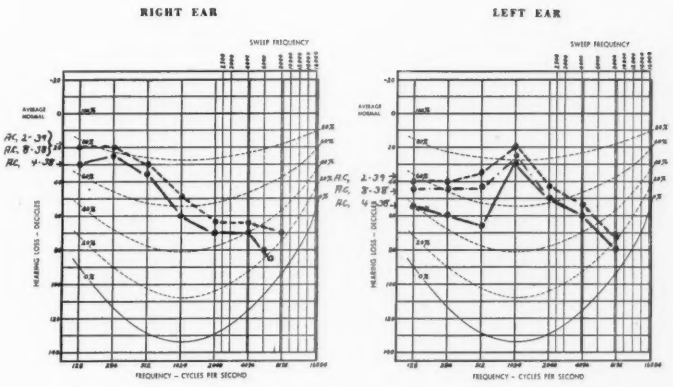

Fig. 6.

CASE 5.—Mr. J. B., age 65. Treatment: May 1, 1938 to August 3, 1938, nicotinic acid, 66 mg. t.i.d. Hearing curve at the end of this time showed a definite improvement, particularly in the left ear. On August 3, 1938, there were added thiamin chloride injections, 3000 I.U. per cc., one cc. daily until November 1, 1938, when the dosage was reduced and continued until February 6, 1939. An audiogram made at this time showed no further change in the right ear, but an additional step-up in the curve of the left ear. Patient's constipation has been relieved and there has been a definite improvement in his general health.

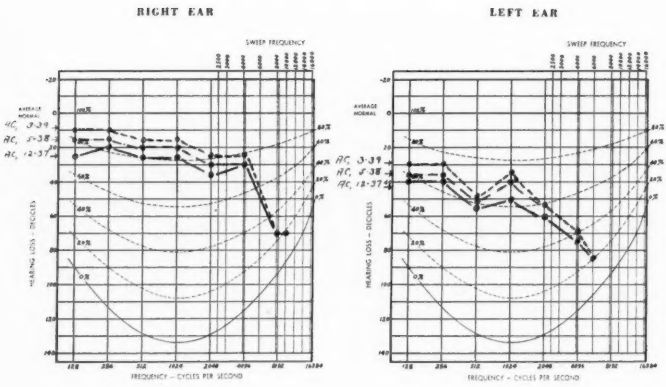

Fig. 7.

CASE 6.—Mr. W. S. B., age 73. The very striking thing about this patient, aside from his loss of hearing, has been a sense of instability on arising in the morning (no real vertigo), his mental confusion, and some loss of his sense of direction. These symptoms have disappeared under treatment. Treatment: December 2, 1937 to December 29, 1937, seven injections of Lederle's B_2 complex; December 29, 1937 to June 13, 1938, nicotinic acid, 66 mg. daily by mouth. March 1, 1938 to March 29, 1938, nine injections riboflavin (Hoffman-LaRoche) in addition to nicotinic acid capsules. Hearing curve following the use of these materials showed a slight gain in both ears. On May 5, 1938, a series of ten injections of thiamin chloride was started (3000 I.U. per cc.). On June 13, 1938, the dosage of the nicotinic acid capsules was increased to 66 mg. t.i.d., and the injections discontinued. There was no change in hearing at this time. Another audiogram taken on March 14, 1939, showed an additional improvement in the curve.

CONCLUSIONS

It appears from the foregoing that thiamin chloride was an essential addition to nicotinic acid for the increase in the hearing curve, as shown in the various audiograms of cases herewith submitted. It is evident from the study of all our charts that the greatest improvement comes from the use of nicotinic acid or sodium nicotinate, and only rarely is the greatest improvement due to thiamin chloride, rather than nicotinic acid. Without doubt, if and when vitamin B_6 is available in the synthetic form, it will prove of great value, as will also Gyorgy's[11] cirrhosis producing factor that is essential as a liver damage protector, especially in cases consuming too much alcohol. His factor is different from B_6, riboflavin and B_1. Morgan's gray hair factor is equally important, as is also Jukes' chick dermatitis factor.

It also goes without saying that any obvious dysfunction of the endocrine glands (pituitary, thyroid, gonads, etc.) should be taken care of as also other vitamin deficiencies, and any abnormal level of the electrolytes, and any lowering of protein intake. Obviously the dietary intake of essential foods should be kept up at an optimum level, especially during pregnancy, infancy, childhood and adolescence. Finally, the data herewith submitted suggests strongly that nutritional deficiencies offer the best solution for the degenerative process involving the auditory nerve.

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XXXV

X-RAY TREATMENT OF INFECTIONS: A REVIEW OF THE LITERATURE AND REPORT OF CASES OF MASTOIDITIS AND SINUSITIS*

B. R. DYSART, M.D.

PASADENA

About five years ago we noticed a decided clinical improvement in a number of cases of acute mastoiditis following the taking of x-ray pictures for diagnosis. On one occasion, two children in the same family developed mastoiditis during measles. The organism was staphylococcus in both cases. X-rays showed very cloudy mastoids; there was acute tenderness on pressure over the whole mastoid, and profuse purulent discharge in both cases. A consultation was held after obtaining x-ray pictures, and operation advised. During the night, after irradiation, temperature came down to normal, the children slept without pain for the first time in two weeks, and on the next day tenderness of the mastoids in both cases was much diminished. The operations were postponed and within five days discharge stopped. Both cases recovered without operation. I remember making the remark to the mother that, if I had not known better, I would have thought that taking the x-ray pictures had cured the children.

Shortly afterward, the son of a physician friend of ours showed immediate improvement following x-ray pictures taken of a scarlet fever mastoiditis. Operation had been considered but he cleared up without this procedure.

On talking the matter over with Dr. Carl Parker, our roentgenologist, we found that there had appeared in "Radiology" an article by Dr. William L. Ross[1] of Omaha on the treatment of mastoiditis with x-ray. He had treated forty-one cases during seventeen years with recovery without operation. We then began cautiously to treat mastoiditis cases which seemed so severe that they might

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*Accepted as candidate's thesis for entrance into the American Laryngological, Rhinological and Otological Society.

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come to operation. We have not run a control series, but have used our treatment on any case that was considered serious. Twelve cases of acute mastoiditis, thirty-one cases of sinusitis in different stages, and eleven miscellaneous cases, including furuncle of the nose and ear canal, have been treated in the last year, reports of some of which are given later.

We have just finished a review of the literature on x-ray treatment of inflammatory process of all kinds, and over two hundred articles were found. No one could read these articles without becoming convinced of the good effects of this method of treatment.

Arthur U. Desjardins,[2, 3] of the Mayo Clinic, quoted ninety-eight articles on x-ray treatment of inflammatory processes; sixty-one of these articles appeared in foreign publications. In part, Dr. Desjardins' article states:

"The value of radiotherapy in the treatment of many acute, subacute and chronic inflammatory processes is not as well known as it deserves to be. This is apparently because the sound experimental basis and the mass of clinical and other evidence on which it rests have not been considered.

"Evidence of the therapeutic value of irradiation in inflammatory processes is so abundant, and the testimony is so generally favorable, that one wonders why irradiation is not used more than it is."

Small doses are better than large doses; too large doses can aggravate the process instead of reducing it.

"It seems likely that the infecting organism also plays a part in the effect of irradiation. The fact that streptococcus infection often causes little or no leucocytic infiltration may explain why, except in erysipelas, inflammatory lesions of this character do not respond so well, on the whole, as lesions caused by the staphylococcus."

Desjardins has given us the best explanation of the probable mode of action of irradiation for infections:

"In 1903 and 1905 it was proved by Heineke that the effect of radiation in animals was to destroy lymphocytes, leucocytes and eosinophiles. This has been proved by many others, including Warthin. Warthin found unmistakable evidence of disintegration of lymphocytes within fifteen minutes after exposure of animals to x-rays. Similar effects follow radium. It is possible that variation in the leucocytic infiltration in different patients may explain the partial or complete failure of irradiation in some cases.

"But since leucocytic infiltration is such an important factor, why is x-ray destruction good? It seems not unreasonable to deduce that irradiation, by destroying the infiltrating lymphocytes, causes the protective substances (antibodies, etc.) contained by such cells to be liberated and to be made even more readily available for defensive purposes than they were in the intact cells.

"The nerve cells of the brain, spinal cord and peripheral nerves are the most resistant of all tissues in the body to radiation. There is probably no stimulating effect on any cells by irradiation.

"As examples of inflammatory lesions notably amenable to roentgen irradiation may be mentioned furuncle, carbuncle, acute simple adenitis, acute parotitis, complicating operations on the colon and other abdominal structures, abscesses and cellulitis of soft tissues, onychia and paronychia, mastitis, sinusitis and mastoiditis in selected cases and delayed resolution of lobar pneumonia.

"In chronic inflammation the dose must be larger than in cases of acute inflammation. Whereas in acute inflammation the dose may vary from 25 to 50 per cent of the limit of tolerance of the skin, in chronic inflammation the most effective doses vary from between 50 and 80 per cent. Still more important, however, maximal benefit requires that irradiation be repeated a number of times at intervals of three to four weeks. Complete resolution is slow and can be obtained only by repeated treatments. Bazy showed (1928) that large doses of x-rays not only remain ineffectual but may actually increase the inflammatory process."

Numerous experimental articles show no bactericidal or inhibitory effect on organisms in culture media. An experimental study on animals by Soto et al[4] of the University of Chicago produces the following:

In experimental animals, suspension of live organisms were injected by hypodermic in different parts of the body subcutaneously. One area would be left as control and the other area treated by x-ray. Best results were obtained if x-rays were given early, before suppuration had developed. No noticeable effect if treated late.

In 1922, Charles Goosmann[5] stated that he had noticed, in 1919, benefit following x-ray pictures. He subsequently treated ten cases of postoperative mastoiditis with continued suppuration following operation. The period between operation and treatment varying from twenty-five days to five years. All ten cases cleared up rapidly, with relief of pain and improvement of hearing. His dosage was about one-half an erythema dose.

Heidenhain and Fried,[6] after quoting many cases with good results, warned that Wagner had reported three cases with fatal results following use of heavy doses in purulent pelvic infections. Small doses had given no bad results.

Friedman[7] of Brooklyn treated 1,018 cases of superficial inflammatory diseases, including 21 mastoids, with good results.

L. S. McAlister[8] of Muskogee, Oklahoma, states that diphtheria carriers responded well to x-ray treatment in dosages of 75 r. to 200 r.

Amedee Granger[9] of New Orleans reports:

"In a number of cases too large (more than 30 cases) to be attributed to mere hazard or coincidence, patients with definite signs of infection and occlusion, but not of softening and destruction of the mastoids, were decidedly improved by the irradiation incidental to the making of the radiographs and went on to resolution and cure."

Technique: Exact dose not given but apparently very little more than a picture dose. In a number of cases with bilateral mastoiditis, one treated ear healed rapidly while the untreated ear persisted. "A constant reliable indication that the diseased mastoid is responding favorably is for a slight general reaction—a rise of one-half to one degree temperature and local reaction, diminished pain and more profuse discharge, to take place in from six to twenty-four hours after treatment, followed the next twenty-four hours by a diminution in the quantity, or a change in the quality—thinner and less purulent—of the discharge, or both."

An excellent paper by Raphael Schillinger,[10] otologist of Brooklyn, states:

"The clinical course of acute mastoiditis appears to take a turn for the better after exposure of the infected mastoid to the roentgen ray. The turn is marked by a syndrome of favorable action, characterized by a drop in temperature, absence of pain and insomnia, diminution in the amount of discharge and a change in the character of the discharge from purulent to mucopurulent. The use of irradiation on cases of acute mastoiditis in which operation is indicated will, in the large majority, cause resolution and cure without operation."

A series of 38 cases, sufficiently advanced to justify operation, were treated and recovered without operation. While treating this series, seven cases developed symptoms sufficiently severe to make operation necessary.

Careful and complete charts are given of the symptoms and treatment of each case. A careful paper. Most cases received only enough x-ray for two plates of each mastoid. A few later cases received "therapy" doses, about 25 per cent of an erythema dose.

W. Warner Watkins[11] published a complete and careful resume on x-ray treatment of inflammations. Two hundred and twenty-three articles were listed and many of the important articles were abstracted. He has this to say on x-ray treatment of infections:

"The evidence has been accumulating for more than 30 years that roentgen therapy is valuable in a great variety of inflammatory lesions. The chronological review of the most important articles appearing in the Journals since 1905 is an almost dramatic demonstration of the tardiness with which the medical mind accepts new ideas.

"Carbuncles: In 1906, Coyle reported the successful treatment of carbuncles by x-ray. Dunham, in 1916, reported the successful treatment of sixty-seven consecutive cases. In spite of this, seven years passed before the next report appears, this time a surgeon, Lewis, in 1923, reported sixteen cases, only two of which required any surgery whatever. In 1924, Hodges reported treatment of nine

large sloughing carbuncles, in all of whom pain was relieved in from six to twenty-four hours and all healed." He concluded, "Every carbuncle should have x-ray treatment as soon as diagnosed."

In 1924, G. C. Andrews, a surgeon, said, "Roentgen therapy is almost a specific in lesions of the superficial type, causing prompt relief of pain, softening and evacuation of pus and rapid recovery, and even in the deep type, roentgen therapy is always of some benfit."

Further quoting Watkins, he states regarding sinusitis:

"The writer's attention was first called to the beneficial effect of roentgen irradiation in sinusitis by a rhinologist who suffered from a chronic infection of the nasal accessory sinuses. He came at frequent intervals to have a complete set of sinus pictures made, and finally volunteered the information that he had the pictures taken for their therapeutic effect. He had found from experience that the amount of exposure required to make six films, necessary for adequate examination, was just the amount of radiation he needed for relief of symptoms for about two months. He stated that he had long since learned never to explore the antrum until x-ray treatment had been tried and failed, and he seldom found it necessary to invade the antrum either through the natural or an artificial opening."

Several series of treatment of pneumonia with good results have been reported:

McIntire and Smith[12] of Texas report thirty-four cases; and Eugene Powell of Temple, Texas, reports one hundred and three cases. Powell[13] states that pneumonias caused by the type III pneumococcus responded quickly and favorably. His mortality in one hundred and three consecutive cases was five per cent.

On bronchiectasis, Maurice Berck and William Harris[14] of Mt. Sinai Hospital, New York, report that previous treatment at Mt. Sinai of bronchiectasis with small doses produced no effect. Heavy doses, averaging 1200 r through each of the anterior, lateral and posterior fields, crossfired, produced remarkable results. Improvement was slow, requiring four months for full benefit of treatment. Thirty cases were treated, with 50 per cent greatly improved or cured. Some severe cases were reported in which the sputum would be reduced from sixteen ounces per day to one ounce per day, with complete loss of foul odor.

Most of the articles give dosages in percentages of erythema doses. (An erythema dose is that quantity of radiation which, when delivered at one sitting will produce in at least 95 per cent of cases a definite reddening of the skin within one week followed by bronzing, without permanent injury to the skin. An erythema dose is equal to

350 r unfiltered, while it is equal to about 450 r if filtered with four millimeters of aluminum.)

In the last few years the accepted unit of dosage is the "r." According to Kaplan:[15] "r equals that quantity of irradiation which, when the secondary electrons are fully utilized and the wall effect of the chamber avoided, produces in one cc. of atmospheric air at 0° centigrade, and 76 cm. of mercury pressure, such a degree of conductivity that one electrostatic unit of charge is measured at saturation current."

Most roentgenologists feel that the picture dose is inadequate except in rare cases. It is probable that the picture dose is not more than 5 or 10 r, and very few roentgenologists advise smaller than 50 r for any treatment. In the acute sinusitis cases without drainage, in which there is pain, I feel that the dosage should be quite small. Some of the acute frontal cases complained of increase in pain in the first few hours, but were relieved on the day following.

The average dose measured by "r" for early cases is considered to be 50 to 60 r; for the more chronic cases, doses as high as 100 r or more are used. Measured by the erythema method, 25 to 50 per cent of an erythema dose has been advised in the early acute cases, and as high as 80 per cent of an erythema dose for the chronic cases.

Twelve cases of acute mastoiditis, in which x-ray pictures showed sufficient haziness to consider operation, were treated by small doses of x-ray, from one to four doses being given at intervals of about five days. All cases cleared up without mastoidectomy. During this time a number of cases were seen after the case had progressed to necrosis. In these cases x-ray treatment was not tried.

REPORT OF CASES

CASE 1.—Boy, aged 4½ years, with a green streptococcus infection. Drum incised on the day of onset, January 8, 1937. On January 31, the x-ray showed cloudy mastoid, no necrosis. There had been profuse drainage with temperature to 101 at intervals since incision. One x-ray treatment of 50 r was given January 31. Ear dried four days after treatment. Drum healed without perforation and with no loss of hearing.

CASE 2.—Boy, aged 12 years, seen January 26, 1937, with temperature of 103.4. Drum incised. Incision of drum was followed by profuse purulent discharge with mastoid tenderness. X-ray pictures taken four days later showed cloudiness throughout left mastoid but no necrosis. X-ray treatment given same day. Moderate discharge without tenderness persisted. Second x-ray treatment was given eight days later; discharge stopped entirely two days later. Dosage in r not calculated.

CASE 3.—Woman, aged 46 years, with staphylococcus and nonhemolytic streptococcus infection. Drum incised two days after onset of pain, April 13, 1937. Discharge was profuse and there was some tenderness of the mastoid. X-ray taken seven days later showed moderate cloudiness of the mastoid. Two x-ray treatments were given, one four days after incision and the second three days later. The ear was dry two weeks after onset.

CASE 4.—Woman, aged 40 years, with bilateral otitis media following cold. Left drum incised a few hours after onset of pain on the respective sides. Mastoids very tender, very high fever and drainage very profuse. X-ray treatments were given early, on the third, fifth and seventh days after onset. Eighty r given on each treatment. Left ear became dry within six days and the right within eight days.

CASE 5.—Girl, aged 5½ years, with hemolytic streptococcus and staphylococcus infection. Drums incised by pediatrician on January 10, 1938. Seen first by myself on February 4, 1938. Both canals were filled with pulsating, thick discharge; mastoids were tender. There had been moderate intermittent temperature. Left mastoid cloudy with no necrosis. Given three x-ray treatments of 50 r, 50 r and 60 r, on February 4, 7 and 11. Sulfanilamide was also started, 2½ gr. q.4.h. The ears were almost dry in two days and completely dry in eight days.

CASE 6.—A boy, aged 13 years, was first seen August 3, 1938, giving history of acute pain in the left ear for twelve hours following swimming. Drum was incised at the first visit and very profuse bloody pulsating discharge continued for six days. Culture showed staphylococcus with a few anhemolytic streptococcus. X-rays taken at that time showed mastoid very cloudy throughout with no necrosis. Treatment of 65 r was given. Next day, following treatment, there was less discharge and less tenderness and patient looked generally better. X-ray treatment was given again in two days with 82 r; again in three days with 82 r and again in six days with 82 r. The discharge stopped completely by the end of the third week, and at the same time the mother said that his sinuses were better than for many years. Child had had chronic sinus trouble, and window openings had been done by a specialist in Los Angeles with very little help. He had continually had purulent discharge from the nose. Improvement in the condition of his nose was very marked. A few additional treatments were then given to the sinuses with further improvement.

CASE 7.—(One other ear case is included, although the duration was so short it must be considered an otitis media.) A football coach from the Middle West reported shortly after our New Year's Day game, with pain in the left ear, temperature 101 degrees, which he had had for about five hours. Drum was incised; after incision there was profuse bloody, pulsating discharge which required changing of the cotton wick every fifteen minutes. He was advised, therefore to have x-ray treatment, as he wished to return to his home as soon as possible. It was explained to him that the probable duration of the infection would be around ten days without x-ray treatment, and possibly much shorter with treatment. On the day following incision and x-ray treatment, we were much surprised to find the ear almost dry and the next day completely dry, with healing of the drum.

On the whole, x-ray treatment of sinusitis has not been so satisfactory as the treatment of mastoiditis and otitis media. It has been more difficult to determine whether or not the patient would

have cleared up without the treatment, as it is the rule for acute cases to clear up whether or not treatment is instituted. Another reason for unsatisfactory results in sinusitis cases is that so many of them have become chronic before being seen. A few cases did respond with very satisfactory improvement.

CASE 8.—Boy, aged 17 years, with profuse purulent pansinusitis for many years duration with complete loss of smell. Culture showed anaerobic streptococcus. The upper two-thirds of nose was filled with polypoid granulations. Window openings had been made several years before by myself with moderate benefit. He was advised at that time that further surgery would probably have to be done when he was older. On several occasions numerous polypi were removed from the nose. On one x-ray treatment, June 15, 1937, there was an immediate lessening in the amount of the discharge from the nose, and within three days the patient stated that for the first time in five years he could smell normally. Several other treatments have been given with continued improvement. Mucous mmbrane has changed from granular polypoid appearance to almost normal mucous membrane. Proetz treatments were started at the same time as the x-ray treatments, and it is possible that they are partially responsible for his improvement.

CASE 9.—An automobile dealer, 55 years of age, had a window opening eight years ago in the left antrum for chronic purulent maxillary sinusitis by my associate, Dr. W. H. Wimp. Had frequent attacks of pain before operation, which the window opening relieved; however, he continued to have a moderate purulent, odorless discharge. A smear made showed many pus cells and no eosinophiles. Occasional irrigations during this time never failed to wash out some pus. He had tried a great many treatments by other doctors without any benefit. Had frequent nagging pain in the antral region; culture showed mixed infection with Friedlander's bacillus, and hemolytic and green streptococcus. After two treatments of 60 r and 75 r, he stated that there was no noticeable pus and the pain had disappeared entirely. Several more treatments were given. He then went for three months without symptoms, and then caught a fresh cold and there was again considerable pus in the nose; without, however, so much pain. Two more x-ray treatments were given at that time, again relieving his condition greatly.

CASE 10.—Girl, 13 years of age, came in for stuffy nose, yellow postnasal secretion and bronchial cough with temperature of 100 degrees, following deep diving during the summer. Nasal treatment over a period of two weeks produced very little benefit. X-ray showed cloudiness of most of the sinuses and much thickening of both antra. X-ray treatments of 50 r were given over the sinuses and 70 r over the mediastinum. These treatments brought the temperature to normal with much less secretion in two days. Second treatment of the same length, given six days after the first, cleared up her trouble completely.

CASE 11.—Boy, 10 years of age, came in March 15, 1938, complaining of stuffy nose with much mucopurulent secretion of six weeks duration. Nasal treatment for several days produced no improvement. X-rays showed sinuses very dense throughout. Mild x-ray treatments (50 r) were given. The next day he reported feeling better than for a month, had better breathing space and no secretion. Six days later had treatment of 60 r with almost complete relief. Cleared up completely within the next two weeks.

CASE 12.—Woman, aged 25 years, seen first March 24, 1938. Nose was badly congested, antra rather dark on transillumination. Gave history of having cold for eight days. Was sent for x-ray plates and x-ray treatment. X-ray pictures showed all sinuses hazy, right antrum especially dense. X-ray treatment given of 50 r. Two days later patient came in and reported that her pain had entirely disappeared, no pus in nose, and no treatment was given. Patient was asked to return for another x-ray examination at the end of the week. At that time there was no secretion but x-rays showed very slight thickening of the antrum still remaining. The rest of the sinuses were clear.

Many of our other sinusitis cases are not reported because other methods of treatment were used at the same time. Two of our worst cases of pansinusitis in adults showed no improvement whatever after five or six x-ray treatments, possibly due to their long duration.

I was just informed by Drs. Wells and Ambler of Arcadia, California, that they have been using x-ray treatment in their otitis media cases as soon after onset as possible. In about twenty-five cases that have come to them before the drum had ruptured, they have given one x-ray treatment of 50 r and the infectious processes have subsided without opening of the drum. In only one of these cases was opening of the drum necessary. In all of these cases there was pain, deafness and redness of the drum. Many of them had bulging to the point where incision of the drum would have seemed advisable. In most of these cases the pain subsided in from four to six hours following x-ray treatment. This was their entire series for the winter of 1937-38. Dr. Ambler is a general physician, but being in a small town, he sees many cases of otitis media, there being no specialist in his community. Dr. Wells is a roentgenologist.

Anyone wishing to review the literature further is referred to the articles of Desjardins and Watkins.[2, 3, 11]

CONCLUSIONS

1. A review of the literature on x-ray treatment of inflammatory conditions shows much favorable, and little unfavorable, evidence.

2. Treatment should be started as early as possible.

3. Small doses produce better results than large doses; very large doses may be harmful.

4. Several cases of mastoiditis and sinusitis were treated with good results.

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XXXVI

TINNITUS: ETIOLOGY AND EVALUATION OF THE VARIOUS METHODS OF TREATMENT

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ETIOLOGY

One of the most distressing symptoms encountered in otology is that of head noises. The patient with a persistent tinnitus complains most bitterly of the annoying tones he is obliged to endure. As a rule he does not object so much to the loss of hearing as to the constant head noises, and in the majority of cases the patient is willing to sacrifice his hearing and become totally deafened if, by so doing, he would be relieved of the constant roaring in his head.

Tinnitus includes all general auditory sensations not caused by sounds outside of the body. According to the outline of Koester,[1] these noises may be divided into two distinct groups: (1) The so-called entotic and the periotic noises, and (2) subjective noises. When these noises occur in the ear itself and are caused by an impairment of the auditory conducting apparatus, they are called entotic; when they occur in the vicinity of the ear or are brought to it from elsewhere in the body, they are called periotic, and when they are purely affectations of the sound perception apparatus they are called subjective noises.

It is not difficult to demonstrate the cause for entotic and periotic noises, but no final reliable theory has been satisfactorily proven. In connection with impairment of the sound conducting apparatus, vascular and muscular noises not normally perceived must be reckoned with; thus through an adhesive process, such as acute inflammation of the tympanic cavity, the normal function of the ossicles becomes impaired and vascular and muscle noises become apparent. It so happens that in affections limited to the conducting apparatus

the noises may be either entotic, or periotic, according to circumstances.

Subjective noises are much more difficult to explain, as they arise in the sound perception apparatus, and are due to mechanical stimulation of the acoustic terminal apparatus or an involvement of the auditory nerve itself through a hyperemia, vascular distention or changes in the labyrinthine pressure. According to the now accepted theories, these subjective noises are due to an irritation or degeneration in the peripheral neurones of the auditory nerve (Hegener[2]), which may be either an acutely inflamed condition or a progressive atrophy (Hegener and Kobrak[3]). These subjective noises will be perceived even after the cochlea has been destroyed or the nerve severed.

Diseases in which subjective noises may occur are chiefly obstructions in the external auditory canals, inflammations of the middle ear, hemorrhages in the middle ear and diseases of the large blood vessels, acute and chronic suppurative otitis media. A large number of diseases showing irritation or degeneration changes in the nerve endings of the auditory perceiving apparatus produce subjective tinnitus. These chiefly are general vascular impairment (arteriosclerosis); pernicious and secondary anemia; typhus, pneumonia, meningitis, osteomyelitis, leukemia, tuberculosis, variola, varicella, grippe; poisoning due to quinine, arsenic, phosphorus, iodine, salicylic acid, alcohol, tobacco; endocrine derangements and disordered metabolism (diabetes, Basedow's disease); tumors and cysts; skull injuries, fractures and concussions; old age auditory difficulties (Edinger theory of too great demand); noisy trades, as forge work, locksmithing, artillery fire, etc.; otosclerosis; juvenile progressive hearing difficulties; concussion of the labyrinth; labyrinth inflammations; Ménière's disease; general mental and physical exhaustions and neurasthenia. In the majority of these ailments, histological changes may be found in the auditory nerve and its terminations.

Tinnitus and deafness are usually parallel. Cases, however, are encountered without head noises but with impaired hearing and vice versa. Frequently the ear noises are the initial symptoms and deafness develops later. The age factor is a predisposing cause of tinnitus in older persons because of arteriosclerosis, vascular changes and metabolic disturbances. Stein's[4] survey of 500 persons with ear noises disclosed 117 had purely middle ear disease, 37 had otosclerosis, 111 had middle ear infections, 230 had inner ear disease; of the 500, 64 per cent had circulatory disorders, and five per cent had no pathological changes.

Noises in the ear which can only be heard if the external noises are excluded are probably caused by inadequate stimulation of the cochlear apparatus. If the external auditory meatus is closed by some foreign body, three types of tinnitus can be heard, namely, extremely low cardiac sounds, hissing sounds that synchronize with the cardiac systole, and weak, almost constant buzzing and roaring noises. The two latter types of noises are caused by the mechanical pressure exerted by the foreign body in the canal but are too weak to drown out the physiologic noises. The hissing noises that synchronize with the cardiac systole are due to arterial pressure, and the continuous buzzing and roaring noises are the result of pressure from the arterioles, capillaries and veins.

EVALUATION OF METHODS OF TREATMENT

If the therapy of a problem so enormous and so baffling is to be at all effective, it becomes apparent that the underlying cause or causes must be thoroughly and systematically studied. Frequently we are inclined to look upon the tinnitus as associated with some ear condition and forget that it may occur either independent of an ear disease or may be aggravated by some underlying general condition.

The following examinations are essential: (1) Complete physical and laboratory examination, including a careful history; (2) otolaryngological; (3) neurological, since the association of tinnitus with some intracranial disease must always be borne in mind; (4) dental examination, not merely searching for abscessed or carious teeth or unerupted molars, but what appears to be a very common condition: some abnormality of the bite. In Goodfriend's[5] series, 55 per cent of an average group of men between twenty and thirty years of age had abnormal dental occlusions; furthermore, 92 per cent in a series of objectively negative ear cases had abnormal dental occlusions; deafness and tinnitus form the most prevalent symptom-complex associated with bite abnormalities. Finally (5) allergic examination; a number of cases have recently been reported of allergic whistling of the ear induced by different substances.

Also, a careful study must be made of the patient's habits of life and his nervous temperament. All factors having to do with the increase or decrease of the intensity of tinnitus must be considered, and it is advantageous to look upon these factors from a prophylactic point of view. Some of the more important factors are listed as follows:

1. *Factors tending to increase the intensity of tinnitus:* Poor health; poorly ventilated rooms; mental and physical fatigue; various noises; pregnancy, menstruation and menopause; over-indulgence in alcohol, tobacco, drugs; various occupations in which the patient comes in contact with toxic gases or fumes as well as with noisy machinery; rapid changes of atmospheric pressure; climatic changes.

2. *Factors having a tendency towards making tinnitus less noticeable:* Good health; outdoor life; keeping mentally and physically occupied, particularly the latter, as it is known that tinnitus is most troublesome in people doing mental work; constant companionship; hobbies; proper attitude on the part of the physician, which includes a sympathetic understanding of the patient's problems, keeping him from believing that his condition is hopeless, assuring him that the condition is not a forerunner of insanity, as many laymen still believe.

After the underlying disorder has been discovered, the chief attention, of course, should be given to treating it in the most energetic manner possible. However, in many cases the disease is of such a nature that even the best known treatment has little effect on it, or the cure of the disease may be affected and yet absolutely fail to control the remaining tinnitus. In view of these difficulties and in view of the fact that in so many cases the underlying cause cannot be discovered, there developed a large field for the symptomatic treatment of tinnitus. This field includes various medicinal, mechanical, thermal, electrical, psychological, dietetic, roentgenological and surgical procedures. In reviewing the literature one is impressed by the fact that the number of therapeutic measures is large but that their relative value is indeed small.

SYMPTOMATIC TREATMENT

What are some of the medicines employed and which ones have been found most efficacious? Various sedatives, soporifices, antipyretics, iodine preparations, calcium, atropine, vasodilating substances as sodium nitrate, caffein and theobromine, various endocrine extracts, strychnine, dilute hydrobromic acid and even insulin have all been used. One would hardly be justified in mentioning all of these, except that occasionally one of them is used with success, but from the large number of preparations mentioned, one gathers that for the treatment of tinnitus no one truly satisfactory medicinal remedy has as yet been found.

On the European continent the best results seem to have been obtained by Koester[6] with a substance called "lubrocal," which is a combination of potassium bromide and sodium phenyl-ethyl-barbiturate. He believes that in order to combat the subjective murmurs which are due to stimulation in the end apparatus of the nerves or to sound sources within the body, there must first be a sedative action for the general reduction of the nervous tone of the body; for this purpose bromide salts have long been recognized as an effective remedy; but since, in the majority of cases, the tinnitus is accompanied by insomnia induced by the head noises, it becomes necessary to have a systemic treatment which will influence this secondary disturbance. Furthermore, in view of the tendency toward permanency of the tinnitus, it is also necessary that the medication used should be such that it can be given for a considerable length of time without ill effects. Each tablet of lubrocal contains 0.04 grams of a barbituric acid derivative and 0.6 grams of bromide. The dosage and method of administration are given in the final paper.

Recently Leclerc[7] revived an old remedy which at one time was considered almost a specific against ear noises. He reports good results with either the tincture or fluid extract of cimicifuga racemosa. Starting with smaller doses, he gives 15 to 60 drops of the tincture and 30 drops of the fluid extract a day. He concludes, however, that it is rare to get relief in patients in whom the subjective noises have continued for more than two years.

On the American continent McNally[8] and his co-workers in Montreal attacked the problem of subjective tinnitus from another standpoint. Taking cognizance of Gray's theory that the essential causative factor in otosclerosis is a gradually increasing defect of the vasomotor mechanism, which governs the nutrition of the entire structure of the organ of hearing, their experiments were directed toward alteration of the cerebral circulation or cerebral pressure. Experiments have shown that the cerebral vessels are under the control of the sympathetic and parasympathetic nervous systems. The sympathetic nervous system carries the vasoconstrictor fibers to the brain and stimulation of this system will cause a constriction of the cerebral vessels and a decrease of the circulation. The parasympathetic nervous system carries the vasodilatory fibers, and stimulation of this system will cause a dilatation of the cerebral vessels with an increase of circulation. They presume that since it is possible to influence the vasomotor mechanism by the use of drugs which act specifically on the sympathetic and parasympathetic nervous systems, it should be possible similarly to influence the cerebral vasomotor

mechanism and circulation. They selected the following drugs for their experiments:

1. Sympathetic
   (a) Stimulant—Ephedrine hydrochloride.
   (b) Depressant—Ergotamine (Gynergen-Sandoz).
2. Parasympathetic
   (a) Stimulant—Mecholyl (Merck).
   (b) Depressant—Bellafoline (Sandoz).

Their experiments showed that the stimulation of the sympathetic or depression of the parasympathetic nervous system had a slightly more beneficial effect upon the tinnitus than did the depression of the sympathetic or stimulation of the parasympathetic; in other words, the best results were obtained with the use of such drugs as ephedrine and bellafoline. To further accentuate the vasoconstrictor effect, they used a combination of these two with even more beneficial results. The ephedrine hydrochloride was used in doses of one-half grain by mouth, a total of about one and one-half grains being given daily over a period of two weeks. The bellafoline was given in doses of 0.25 milligrams up to one or two milligrams per day for a similar period. The drugs were also employed hypodermically, but the results were not as good as by the oral administration.

It is interesting to note here that many others recommend a vasodilatory drug like sodium nitrate, particularly the Japanese,[9] who report very favorable results in 84 per cent of their cases.

Another interesting medicinal agent for the treatment of tinnitus was studied by Lewy[10] following an observation by Barany that injection of procaine into the inferior turbinate had occasionally stopped tinnitus. Lewy tried intravenous injections of procaine, nupercaine and quinine hydrochloride with ethyl carbamate. Seventy-five subjects were studied, some over a period of seven months. Of these, the procaine treatment was effective in reducing tinnitus in the majority of cases. The solution used was a one per cent solution, and from seven to ten cubic centimeters of this was given intravenously. He assumes the action of procaine to be central.

Polte[7] made an observation that a patient of his recovered completely from tinnitus after being given a submucous injection of novocain for a submucous resection of the septum. He carried out some experiments of injecting novocain under the upper portion of the septal mucosa, and reports good results in the majority of cases.

OTHER METHODS OF TREATMENT

1. *Mechanical and Thermal:* In addition to the large number of medicinal remedies, we have various mechanical and thermal methods of treatment. Most of them depend on their property of inducing hyperemia in the region of the middle ear and labyrinth in as gentle a way as possible. Here we have such procedures as the introduction of liquids and vapors by way of the eustachian tube, lavage with hot or cold water, treatment with hot air, pneumo-massage, massage through the eustachian catheter, vibratory massage, and finally, bombardment of the cochlea and masking methods. The idea of the bombardment of the cochlea is to desensitize the cochlea in certain tonal regions after previously determining the pitch and loudness of the tinnitus. This is a fatigue process and not a lasting one. The other, the masking device, is a harmonic generator producing a complex spectrum of tones, particularly helpful in sleep. Many an individual with tinnitus can gain the same effect by having a loud-ticking alarm clock close to his ear during sleep. One of our patients would get relief in a factory where there was great mechanical activity. It is not unreasonable to assume that some adequate and comfortable noise apparatus, adjustable in pitch and intensity, may some day be devised to meet the various cases of tinnitus.

2. *Electrical:* A number of cures were reported by the use of various electrical methods. The purpose of all these is the introduction of the electrical current to the tympanum, stapes and inner ear. European otologists are particularly fond of galvanism, although diathermy, both the long and short, faradism, and even a new, complicated procedure called rhythmic galvano-faradization[12] are all employed. This latter method is intended to produce in the muscle of the malleus (tensor tympani) a succession of contractions and relaxations. A displacement of the malleus is communicated to the stapes as an inverse displacement and the force transmitted is greater than that originally produced in the malleus. This series of successive contractions and relaxations in the malleus causes the stapes to exercise a series of compressions and decompressions of the oval window. This in turn reduces the ankylosis of the stapes which, as the author claims, is the most common cause of deafness and tinnitus.

Here in America the results obtained with electrical procedures do not seem to be so startling. In connection with this it may be interesting to relate a recent personal experience. A patient with unilateral chronic catarrhal otitis media had an annoying tinnitus. Under short wave therapy the tinnitus became much less noticeable.

The patient volunteered the information that the pitch of the tinnitus changed from a high to a low tone which he did not particularly mind. Cases are even reported as being cured by exposure to light rays, such as the mercury lamp.

3. *Psychological:* An interesting form of treatment is the psychological. Kümmel maintains that the patients should be instructed concerning the unreality of their murmurs and be given careful psychological treatment. They should be told that the pathology of the situation lies not in the murmurs but in the capacity for hearing them. Good results should be obtained in those cases where the cause of tinnitus is relieved but the tinnitus itself becomes a fixed idea, the so-called "memory tinnitus" of some authors.

4. *Dietetic:* A case is reported here and there of the cure of tinnitus by restricting salt in the patient's diet. Furstenberg[13] and his co-workers reported the results of a very thorough investigation of six cases of Ménière's syndrome in which the metabolism of sodium and water were carefully controlled. They found that the symptoms of Ménière's syndrome are due to the retention of sodium by the body, and by preventing an accumulation of sodium by the administration of ammonium chloride, they were able to keep these patients free from attacks. On the other hand, they were able to precipitate an attack by the administration of sodium. Unfortunately, no mention was made of what happened to the tinnitus in these cases, although it is known that tinnitus is invariably an accompaniment of Ménière's syndrome. On the strength of this report, McNally tried the same technique as outlined by Fuerstenberg. He was forced to conclude that the ordinary case of tinnitus is not benefited by this dietary regime, but did not have sufficient data to say what the effect was on the tinnitus in a definite Ménière's case.

5. *Roentgen Rays:* The use of roentgen ray and radium in the treatment of tinnitus, although again revived at the last Congress of Radiology in Chicago, does not appear to be attended by any definitely successful results.

This brings us to what is known as the "last resort" treatment of tinnitus, namely, surgery. Before we go into the more formidable surgical procedures, mention should be made of the minor ones, such as lumbar puncture, injection of the sphenopalatine ganglion, and vascular ligations of the carotid artery or jugular vein.

Ruskin, in 1920, first recommended injection of the ganglion as a possible cure for tinnitus. Here and there in the literature one

reads of a case relieved by this method. McNally and his co-workers injected the ganglion in thirteen cases with no beneficial results. In one case the noise was made worse, and in another the noise completely disappeared for only about fifteen minutes.

Shigov writes that lumbar puncture proved advantageous in his treatment of 160 cases, the tinnitus becoming less intense.

Iglauer,[14] after reporting the various successes and failures of vascular ligation of the carotid artery and jugular vein, made the following suggestions in his conclusion: If the patient has been greatly harrassed by the tinnitus for a long period of time and if the sounds disappear on light digital pressure of the internal jugular vein as well as after rotation of the neck, the vein might be exposed, preferably under a local anesthetic, and provisionally occluded. If the tinnitus ceases, the jugular vein should be ligated. In this connection it might be interesting to relate the experience of Bodeaux and Reickert, who report the case of a young woman suffering from an annoying tinnitus in the right ear following several cranial traumas. Rotation of the head to the right caused a momentary relief, and the tinnitus disappeared on compression of the right carotid artery. This seemed like the proper case for a vascular ligation. However, while still under observation, the tinnitus became more intense and there developed a weakness of the right fifth nerve. An arterio-venous fistula or tumor was suspected. At operation, a vascular diaphragm blocking the aqueduct of Sylvius was found, coagulated and divided, releasing the obstructed ventricular fluid. The tinnitus disappeared. It was due to an angulation of the right cerebral vessels. An exostosis of the occipital bone pressing against the right trigeminal explained the weakness of that nerve.

Various cervical sympathetic operations for the relief of tinnitus are reported in the literature, many with excellent results, although Brunetti[15] states that in his experience he could not think of a single case in which he had obtained satisfactory benefit as far as head noises were concerned. McNally[16] reported improvement in the tinnitus in three out of four cases following a stellate ganglionectomy (first dorsal sympathetic). He was rather enthusiastic about this method of depression or elimination of the sympathetic, and it is interesting to note that this was contrary to the results that he had obtained with ephedrine, which stimulates the sympathetic nervous system.

We have gone through the stages where opening of the labyrinth, obliteration of the cochlea, severing of the auditory nerve were

all tried and discarded as not only untrustworthy but dangerous. Krause, in 1903, introduced the surgery of the trunk of the eighth nerve or section of one of its branches in cases of persistent noises. Since that period many operators improved on the original technique, tremendously reducing the mortality which at one time was as high as 80 to 85 per cent. The most recent and most brilliant work along this line is that reported by Dandy[17] in 1934 of his treatment of Ménière's disease by total or partial section of the auditory nerve. In his forty-two cases there has been no known attack subsequent to the operation. However, concerning the tinnitus, he draws the following conclusions: It is evident that one can never promise a patient with Ménière's disease that relief or cure of tinnitus will follow the operative procedure. The chances of total disappearance are about 50 per cent. Twenty out of his forty-two cases were completely relieved of the tinnitus. These statistics were recently verified in a personal communication with Dandy.

COMMENT

One cannot help but agree with Brunetti, who made the following conclusion just one year ago after reviewing his own work and the literature on the intracranial section of the eighth nerve: the cases of tinnitus showing clear and unequivocal indications for surgical intervention are indeed rare and he would still much prefer to trust to the conservative measures for the relief of tinnitus.

CONCLUSIONS

1. Tinnitus is unquestionably one of the most aggravating symptoms we as physicians have to cope with.

2. Its causes are numerous and may require the services of an internist, otolaryngologist, neurologist and even a dental orthopedist.

3. When called upon to treat a case of tinnitus, it should be tagged as "curable" or "incurable."

4. If curable, the physician should devote himself systematically toward effecting a complete cure and not merely toward the application of symptomatic therapeutics.

5. If incurable, every means at our command must be exhausted in attempting to give the patient as much relief as possible.

6. The perseverance on the part of the physician in the use of the various measures discussed will give the afflicted one courage to carry on.

7. A defeatest attitude on our part will lead the patient to despair, if not to actual suicide.

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XXXVII

THE INCEPTION AND DEVELOPMENT OF PERORAL ENDOSCOPY

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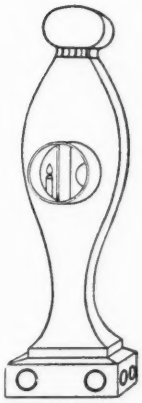
For our purposes, "Seeing is Believing," might be paraphrased "Seeing is Knowing." In medicine, as in other branches of science, man's desire to explore the unknown has led him to replace the element of chance, as represented by guessing, inference and deduction, with accurate information gained by exact visual impression.

The scope of this paper must, of necessity, be limited to a record of the early efforts of those who pioneered in this branch of medicine. Through the years the contributions of hundreds of workers throughout the world has brought peroral endoscopy to its present state of practicability.

The passage of a tube into the esophagus or stomach was inspired by the sword swallowers, while laryngoscopy, tracheoscopy and bronchoscopy were inspired by the laryngeal mirror, and intubation or catheterism of the larynx and trachea. Kussmaul carried out some of his earliest experiments upon sword swallowers. Hippocrates first suggested intubation of the larynx and trachea in cases of impending asphyxia. This procedure was further emphasized by Desault[5] in 1801, when he erroneously passed a feeding tube into the trachea of a patient. As a result of this accident, he was able to demonstrate that the trachea would tolerate a tube for a more or less indefinite period of time. In 1858, Bennett of Edinburgh described the introduction of catheters into the bronchi of seven patients. He injected the lung eleven times and used as much as one-half ounce of an eight per cent solution of silver nitrate. Two years later, Horace Greene wrote a paper on the "difficulty and advantage of catheterism of the air passages in diseases of the chest." This embodied the work of several years and included the treatment of one hundred and six patients.

Peroral endoscopy is, without doubt, the most spectacular and valuable of all endoscopic procedures. It includes esophagoscopy and gastroscopy, and laryngoscopy, tracheoscopy and bronchoscopy.



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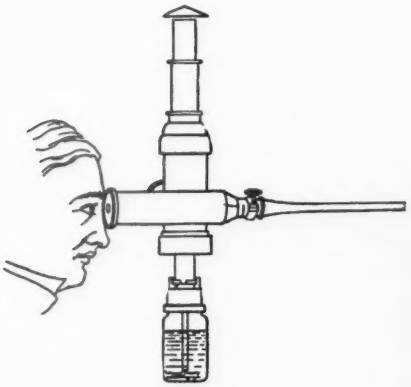

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Fig. 1—Dr. Bozzini's illuminating apparatus. Figs. 2 and 3—Dr. Desormeaux's illuminating apparatus and Dr. Kussmaul's esophagoscope. Fig. 4—Dr. Bevan's esophagus speculum.

One is impressed with the difficulties which the physicians encountered in their cases of foreign bodies in the air and food passages in pre-endoscopic days. Arrowsmith[1] wrote a comprehensive paper on this subject several years ago. As a result of the development of this branch of surgery, the laity have become bronchoscopy-minded.

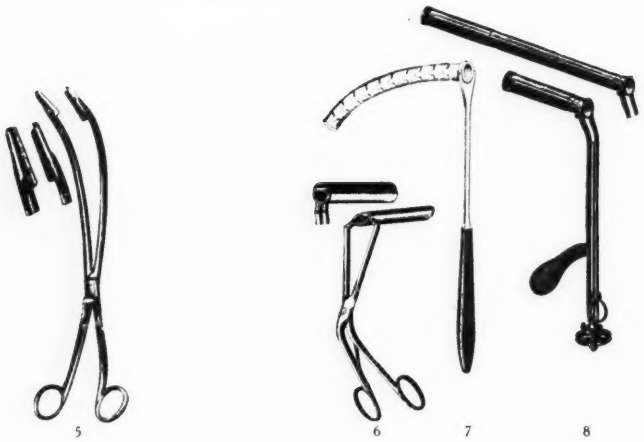

Fig. 5—Doctors Stoerk and Semeleder's esophagus specula. Figs. 6, 7, 8—Esophagoscopes devised by Dr. Stoerk.

Foreign bodies are today encountered much less frequently. In addition, many local diseases are accessible to treatment and amenable to cure which were not recognized in former times.

ESOPHAGOSCOPY

The first attempt to visualize the esophagus is credited to Bozini.[3] He began his experiments in 1795, and some years later reported that he was able to see its upper end. Schindler[27b] mentions that an Englishman named Avery constructed an apparatus during the first half of the nineteenth century, which enabled him to see the urethra, the bladder, the vocal cords and the esophagus. In 1866, Semeleder and Stoerk tried to visualize the upper esophagus by means of a forceps with spoonlike blades, and the laryngeal mirror. They had little success. In 1868, Bevan and Kussmaul, independently of each other, tried to visualize this region by means of a tube, using a mirror at the upper end. In this they failed. Later, Kussmaul investigated the normal and diseased esophagus by the direct method, using the Desormeaux cystoscopic lighting arrangement and a system of rigid tubes. Muller, who was associated with Kussmaul, concluded that a 13 millimeter tube could be passed through any normal esophagus. Killian[16a] discovered and made known the fundamental work of

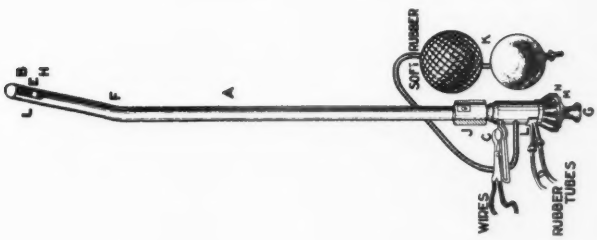

Fig. 9—Dr. von Mickulicz's type of esophagoscope.

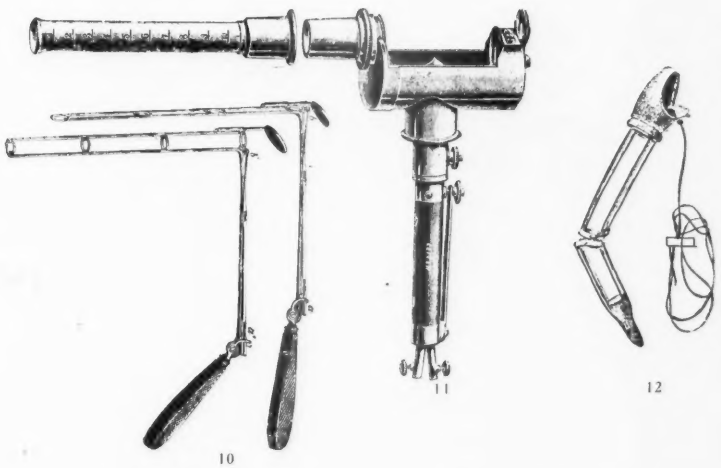

Fig. 10—Dr. McKenzie's esophagoscope. Fig. 11—Dr. von Hacker and Mr. Leiter's pan-electroscope. Fig. 12—Dr. Loewe's esophagoscope.

Kussmaul, who did not publish an account of his studies. Because of this, Kussmaul never received full credit for his efforts.

In 1870, Waldenburg[31] passed an eight centimeter gum elastic tube down the hypopharynx, and with the aid of a mirror was able to diagnose a diverticulum of the upper end of the esophagus. Later he had an instrument constructed of two telescopically arranged tubes, each tube being six centimeters long. In 1881 he introduced a lobster jointed tube covered with India rubber. Stoerk[30b] modified the Waldenburg instrument to facilitate its introduction. He appre-

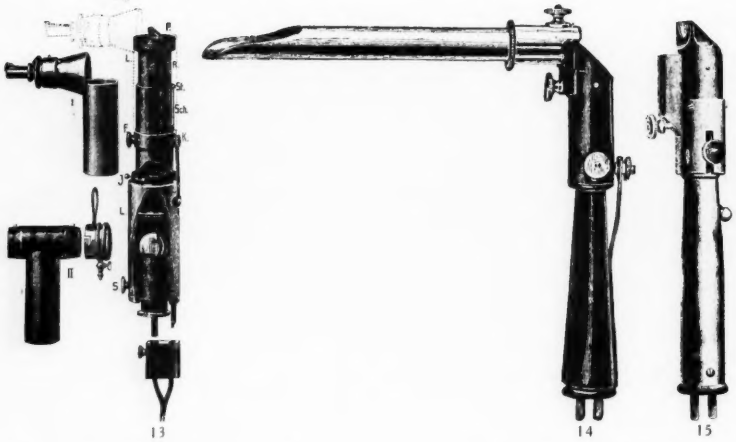

Fig. 13—Dr. Gottstein's electric light concentrator. Fig. 14—Dr. Kirstein's electroscope. Fig. 15—Dr. Kasper's electroscope.

ciated the necessity for longer specula for deeper penetration, which could not be accomplished by means of a latticed tube. As a result he was the first to resort to the use of a long, rigid tube. This was the real beginning of modern, or direct esophagoscopy, as distinguished from the indirect, when a mirror was used. Stoerk was able to make observations on the esophagus and remove several foreign bodies, but he did not publish his experiences. Credit has been given to Mikulicz for the early development and popularization of esophagoscopy. The elder Leiter, an instrument maker, stimulated him to undertake esophagoscopic work without his knowledge of the work of Kussmaul. Stoerk's work, however, was known to Mikulicz.

In the early records of esophagoscopy the efforts of von Hacker, Rosenheim[26] and Gottstein are accorded a prominent place. Kirstein and Killian, however, became the leaders in this field because of their numerous demonstrations and publications.

Killian was the first to introduce a tube without a mandrin or obturator. This afforded the operator a clear visual field so that he could see as he advanced the tube. The elimination of the mandrin marked the first step in the discouragement of blind attempts at extraction and bouginage.

In 1880, Mackenzie[21] continued the attempt to visualize the esophagus by means of a mirror. He used a skeletonized esophago-

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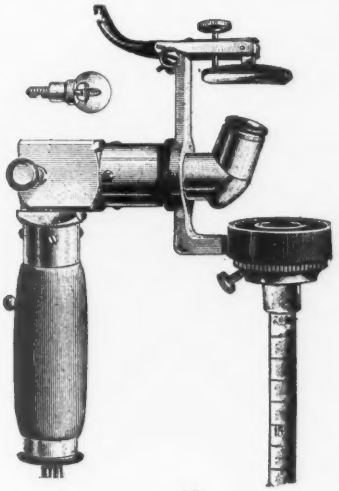

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Fig. 16—Dr. Bruening's electroscope. Fig 17—Dr. Kahler-Leiter pan-electroscope.

scope, which consisted of two wires held together by movable rings. With this he was able to examine 37 out of 50 patients. He successfully removed a foreign body and attacked a carcinoma of the upper esophagus. Jackson mentions that he had the privilege of watching Mackenzie use this instrument in 1890.

In this country, first Einhorn, and later Jackson and Mosher devised esophagoscopes. Mosher's esophagoscope is oval, and is, to this day, preferred in many clinics. His article on the esophagus, which was published in 1906, is fundamental and should be known to all esophagoscopists. In 1911, Richard Lewisohn,[20] New York surgeon, devised a special telescopic esophagoscope. His instrument consisted of two portions joined together at right angles. The distal portion consisted of six tubes which could be passed down the esophagus as far as necessary. Their passage was controlled by a spring connected with them from the proximal rigid end. Illumination was by means of an arrangement similar to that of the Brunings[3] electroscope.

It is interesting to note that the tubes used by Kussmaul and Mikulicz[22] were made of hard rubber, with slanting ends, and furnished with obturators for introduction. According to Mueller,[24]

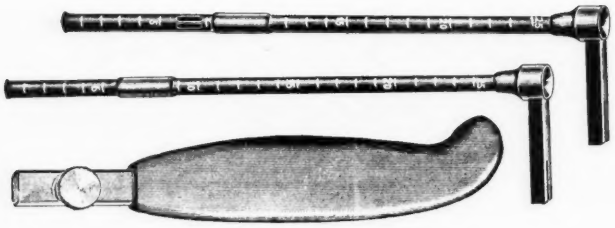

Fig. 18—Dr. Killian's type of esophagoscope and bronchoscope and handle.

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Fig. 19—Dr. Kirstein's electric headlight. Fig. 20—Dr. Clar's electric headlamp.
Fig. 21—Dr. Guisez's electric headlamp.

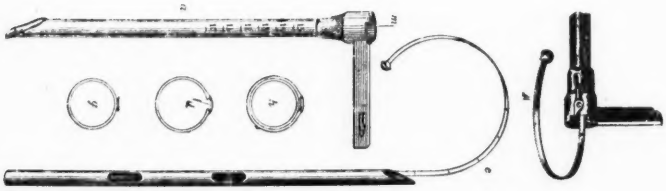

Fig. 22—Dr. Bruening's extension type of esophagoscope and bronchoscope.

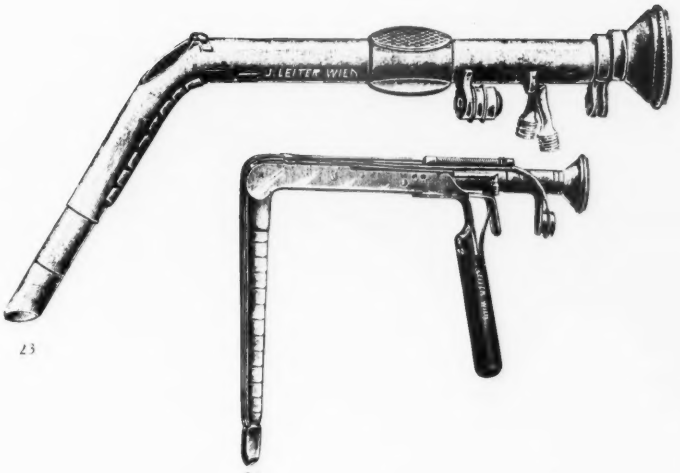

Fig. 23—Mr. Leiter's esophagoscope. Fig. 24—Dr. Nitze's esophagoscope.

it was not until about 1891 that Göttstein[9] used the first boilable tube made of metal.

ILLUMINATION

The necessity for adequate illumination was the stumbling block in all of the earlier experiments. Many attempts were made to furnish illumination for the various instruments which were devised. Most of them proved unsuccessful. It was not until the days of the electric light bulb that practical illumination was available. Bozini described an apparatus for illuminating the cavities of the human body in 1805. His source of light was a candle. About 1820, Desormeaux demonstrated a special gas lamp which he used for illuminating a short urethroscopic tube. In 1881, Leiter[10] suggested a platinum wire burner. This was placed at the distal end of the Mikulicz esophagoscope. It was enclosed in a double walled glass tube through which a constant stream of cold water flowed. This was a distinct improvement over previous methods of illumination, and was replaced by the electric light bulb in 1886.

von Hacker[11] requested Leiter to eliminate the space consuming distal illumination, suggesting that the light be placed in the handle. As a result, Leiter devised his pan-electroscope, which projected

Fig. 25—Dr. Glueckmann's esophagoscope.

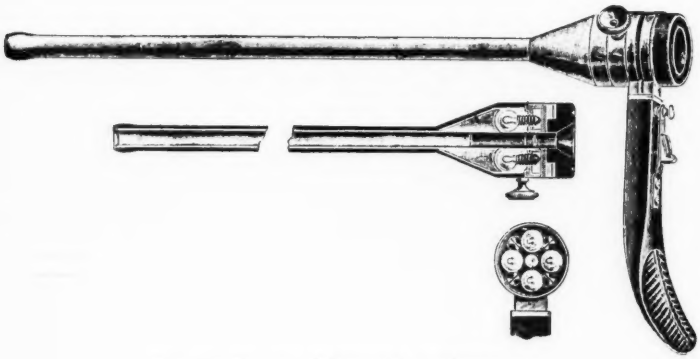

Fig. 26—Dr. von Schroetter's esophagoscope.

parallel rays of light through the lumen of the tube. With this type of illumination the entire lumen was available for visualization and instrumentation. In rapid succession proximal handle lights, or electroscopes were devised by Casper, Leiter, von Hacker, Gottstein and, later, Brunings and Kahler. The Bruning electroscope was first introduced in 1907. It is the best known instrument of its kind and is used in several clinics in this country. This model is considered as nearly perfect as such an apparatus can be made. In all of these, parallel rays of light were projected through the tube from the handle by means of an angulated mirror, Kirstein's[17b] first work was done with illumination furnished by a head-lamp which he devised. Later, Guisez[10] and Clar developed head-lamps for the same purpose.

Glueckmann was the first to use a light carrier within a tube. von Schroetter[20] did not use a light carrier, but used a conical arrange-

Fig. 27—Dr. Lewisohn's telescoping esophagoscope.

ment at the proximal end of his tube in which four small light bulbs were concealed. The light was reflected down through the tube along its inside slanting surface. The inner lining of the tube was made of glass.

In 1902, Max Einhorn[7] introduced the idea of the auxilliary tube in the wall of the esophagoscope for a light carrier. The light carrier had, at its distal end, an incandescent bulb. Subsequently Ingals,[13] Jackson,[14] Mosher[23] and others utilized the Einhorn arrangement for illumination.

GASTROSCOPY

The development of gastroscopy was closely related to that of esophagoscopy. The earliest attempts at gastroscopy were made by Kussmaul in 1868. At about the same time Mikulicz, with the aid of Leiter, produced a rigid tube with a platinum lighting arrangement at its distal end. This was separated from the outside by a window. Two prisms within the tubes formed the basis for the optical apparatus. Nitze and Leiter attempted to construct a gastroscope along the lines of their cystoscope. In 1873, Trouve designed a polyscope which was used by some of his colleagues. Some years later Rosenheim designed and built a gastroscope with optical and lighting arrangements similar to that of Mikulicz.

Jackson used a long tube and the Einhorn lighting arrangement, and was able to report on 70 gastroscopies in 1908.

Fig. 28—Dr. Kussmaul's improved esophagoscope, made of hard rubber.

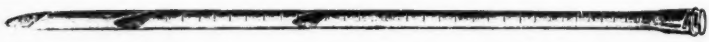

Fig. 29—Dr. Gottstein's esophagoscope, the first tube made of metal.

Fig. 30—Dr. Rosenheim's type of obturator for esophagoscopies.

Fig. 31—Dr. Schreiber's esophagoscope, oval in shape.

Kelling[15] was the first to appreciate the advantage of a flexible tube. A large number of gastroscopes were designed by many different workers. For a complete historical sketch of gastroscopy the reader is referred to the recent textbook on the subject by Schindler. It remained for Schindler, with the aid of Wolf, to create the most practical instrument thus far devised. In 1932 they announced the perfection of a gastroscope which consists of a long, flexible distal segment and a shorter rigid proximal one. A system of lenses furnishes the optical apparatus. It affords a good view of the antral and pyloric regions which were not readily visible with the other instruments. Imperatori[12] has recently been experimenting with visualization not only of the pylorus, but of the duodenum. He calls his procedure "peroral duodenoscopy." The instrument consists of a telescope enclosed in a sheath. It is 64 centimeters long and has a special lense system. The instrument is guided over a thread and is introduced as for esophagoscopy.

Fig. 32—Dr. Jackson's present-day esophagoscope, with two auxiliary tubes, but without obturators.

LARYNGOSCOPY—TRACHEOSCOPY—BRONCHOSCOPY

Attempts to visualize the larynx and trachea were made almost 200 years ago when Levret used a polished metal disc as a mirror with which he attempted to visualize the interior of the larynx. In 1825, John D. Fisher of Boston attempted to perform endoscopy with the aid of candlelight. Two years later Senn made some experiments with a laryngeal mirror, which he published in 1829. His results were of no value. It was not until 1855, when Manuel Garcia introduced indirect laryngoscopy, that this procedure became recognized.

The laryngologists were not content with the exposure afforded by the mirror. Kirstein reasoned that if a rigid tube could be passed into the esophagus, it might also be passed into the larynx and trachea. The fact is that Rosenheim actually passed his esophagoscope into the trachea by mistake, and was able to see the bifurcation. Rosenheim failed to appreciate the significance of his mistake. Kirstein designed a tubular spatula which he called the autoscope. This enabled him to expose the larynx by means of elevation of the epiglottis. He was able to view the passages as far as the bifurcation. He performed his first direct laryngoscopy on April 23, 1895.[17a] He was able to straighten out the curves of the trachea by using tubes designed for esophagoscopy. He was successful in only 25 per cent of his direct laryngoscopies, and partly successful in 50 per cent. Kirstein was afraid to push the tube down into the trachea. In this connection he stated: "The lower half of the trachea is an exceedingly dangerous region. The walls, especially the left, pulsate rhythmic-

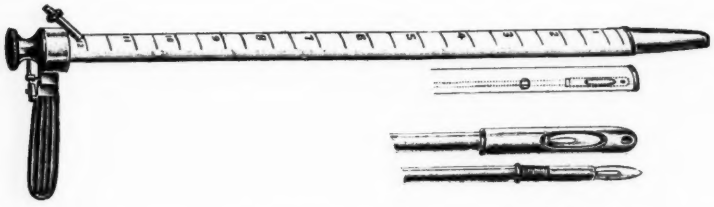

Fig. 33—Dr. Mosher's esophagoscope, oval in shape, with light carrier, light obturator and dilating window.

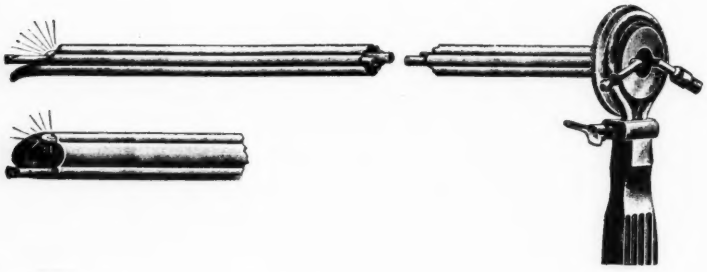

Fig. 34—Dr. Lerche's esophagoscope with light carrier and separate drainage tube.

ally. The pulsation of the aorta can be easily seen, and counted toward the anterior aspect. The pulsation is impressive to watch and calls for the greatest caution in the use of rigid instruments."

A few months later, with the work of Kirstein in mind, and after some preliminary anatomic studies, Killian deliberately passed a nine millimeter tube into the bronchus of a male adult. His assistant, Kollofrath[18] reports how on March 30, 1897, Killian was asked to see a man of 63 who had aspirated a piece of bone into his right main bronchus. After anesthetizing the larynx with a 20 per cent solution of cocain, with the aid of a Kirstein headlight, he passed a Rosenheim esophageal tube into the right main bronchus. He was able to extract the foreign body. This marked the birth of bronchoscopy.

Almost simultaneously with the work of Kirstein, Schroetter and Pieniazek[25] practiced what they termed "Inferior Broncho-

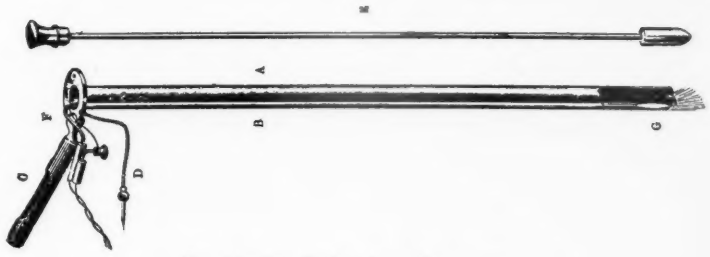

Fig. 35—Dr. Einhorn's esophagoscope.

scopy." This consisted of the passage of a hollow tube into the trachea, through a tracheotomy wound. Schroetter performed inferior bronchoscopy with the patient in the recumbent position and the head hanging over the edge of the table. Pieniazek used both the recumbent and the sitting positions for his examinations. The illumination for his work was furnished by the light reflected from a head mirror. Pieniazek used no anesthesia. He was able to examine children as well as adults. According to him, each bronchial orifice could be visualized if the body were bent away from the structure which he desired to view.

Schroetter improved the illumination for bronchoscopy by introducing the Casper electroscope. Both Schroetter and Pieniazek considered that they were limited in their ability to penetrate beyond the bifurcation. They did, however, pass applicators beyond this region and were able to introduce dilating instruments into both bronchi. Pieniazek later surpassed his colleagues by blindly introducing instruments into the depths of the bronchi and successfully removing foreign bodies. This blind groping further stimulated the desire for visualization.

During his anatomic studies, Killian[16a] was impressed with the thick, hard, elastic, mobile walls of the bronchi. He reasoned that the esophagus was closer to the aorta than the bronchus, and that the bronchus should surely permit the passage of a rigid tube if the esophagus did. With careful preparation and the use of 20 per cent cocain solution for anesthetizing the larynx, he was able to pass his bronchoscope beyond the bifurcation. He was successful without endangering his patient, and felt that the discomfort of which his patients complained was due to pressure against the laryngeal structures rather than the bronchus. At first he used the Kirstein head-

Fig. 36—Dr. Ingall's light carrier with flat stem and flat miniature lamp, for use with Killian tubes.

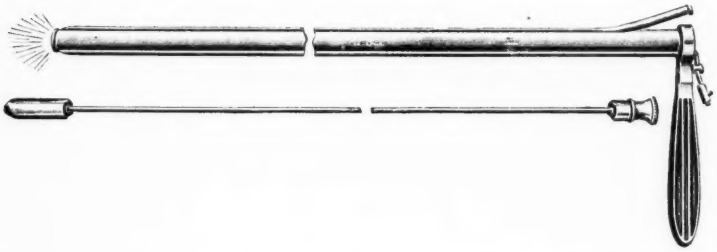

Fig. 37—Dr. Jackson's esophagoscope with two auxiliary tubes, one for the light carrier, the other for drainage and with an obturator.

light for illumination; later he employed the Casper electroscope. There was no resistance to the passage of the tube, no pain, no bleeding and no danger. In the beginning he used a mandrin or stylet to facilitate its passage through the cords. He mentions that in his early work mucus was coughed into his face and also into the tube, from which it had to be removed. His bronchoscopies were limited to patients who were not apprehensive or weak and cachectic. The head was held backward and to the side. He employed a special spatula to push the tongue out of the way, and a hook to push the palate aside and retract the angle of the mouth.

In 1903, von Eicken[6] used, for the first time, a laryngeal speculum through which he guided a longer tube into the trachea. This was the beginning of the technique of bronchoscopy as it is practiced in America by Jackson and his followers.

In 1905, Killian began performing bronchoscopy with the aid of a separable laryngeal speculum. After the bronchoscope was passed, the speculum was separated into halves to facilitate its removal. Jackson modified Killian's speculum by using a removable slide on the laryngoscope. Shortly after, Mosher devised a laryngo-

scope which was hinged on one side. Both of these are used extensively in this country. Laryngoscopes were also devised by Ingals, Yankauer, Arrowsmith, Lynah and others.

Suspension laryngoscopy was introduced by Killian in 1909. This procedure was taken up by many workers. In this country it was used to a great extent by Lockard, Arrowsmith, Freudenthal and Lynch. Lynch modified Killian's apparatus and popularized the procedure.

As esophagoscopy and bronchoscopy developed, new types of tubes were introduced by Killian, Kirstein, Schroetter, Gottstein, Brunings, Guisez, Jackson, Mosher, Ingals, Lynah, and many others. In addition, many instruments were devised for purposes of suction, grasping, cutting, dilating, cauterizing and medicating. A large number of special forceps were designed for foreign body removal.

In this country the first successful bronchoscopy was performed by Coolidge[14a] at the Massachusetts General Hospital. On May 11, 1898, with the aid of ether anesthesia, he extracted the tubular portion of a tracheal cannula from the right main bronchus of a man 23 years of age. He passed a urethroscope one-half inch in diameter and three inches long through the tracheal wound, which he had previously dilated. Perfect illumination was afforded by means of a head mirror which reflected the sunlight. The foreign body was readily removed with a pair of alligator forceps.

Ingals[13] reported the removal of two foreign bodies in 1904. He used the Killian tubes, through which he passed a light carrier for illumination. In the following year Jackson[14a] and Yankauer[32] reported the successful removal of foreign bodies. Jackson reported nine cases which illustrated the difficulties attending foreign body removal in those days. He used Killian tubes, to which he added a special channel for the Einhorn type of light carrier. He also changed the handle somewhat. Yankauer removed an orange pit from the right main bronchus of a ten-months-old infant through a tracheotomy wound. For this purpose he used duplicates of Killian's tubes and forceps which he himself made. In the transactions of the American Laryngological Association for 1904, Hubbard mentions that A. V. L. Brokaw of St. Louis reported, in February, 1903, the removal of a tack from the right main bronchus through a tracheal opening by means of a cystoscope and forceps.

It is interesting to note that Ingals and Jackson used an obturator in their bronchoscopes in these early days. They introduced the

tube with the guidance of the index finger of the left hand, similar to the technique of passage of the O'Dwyer intubation tubes. The obturator was removed by an assistant as soon as the tube passed the epiglottis.

Since the early days of bronchoscopy in America, Jackson has made a steady succession of brilliant contributions to peroral endoscopy. These have stamped him as a master and have made this country the foremost bronchoscopic center. The technique of bronchoesophagoscopy has been spread throughout the world by his teachings. The present high state of perfection of bronchoscopy constittutes a monument to his skill, energy and unusual teaching ability.

The pioneer work of Mosher in the field of esophagoscopy must be given special mention. His original work on the esophagus began with a paper which was published in 1906, and has continued to the present time. It would be of great advantage to the esophagoscopist to study the contributions of this master in our field.

The endoscopists at the Mayo Clinic, under Plummer, Vinson and Moersch, and those in Philadelphia, under Jackson, Tucker and Clerf, have contributed greatly to the field of esophagoscopy and to medical bronchoscopy. Yankauer, Lynah and Imperatori also were pioneers in this field.

It is impossible to give due credit to many workers, especially our contemporaries in this country and abroad, who have contributed much to the development of peroral endoscopy. The roster of the American Bronchoscopic Society, since its inception in 1917, contains the names of many whose work has proven that "Seeing is Knowing."

136 E. 64TH STREET.

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XXXVIII

THE STORY OF PLASTIC SURGERY

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PHILADELPHIA

Plastic surgery was conceived in sympathy and born of necessity.

The Ebers papyrus acquired by George Ebers while excavating at Thebes in 1872, contained extensive references to surgical instruments and plastic surgery. The period is uncertain but contained material copied from older authors estimated to have been written about 3500 B. C. There are described operations for mutilated or missing noses and ears. Apparently such operations were described as being matter of general knowledge rather than objects of wonder. Ancient Hindu literature has come down to us in the Vedas or holy books whose estimated antiquity varies from 2000 to 3500 B. C. At that period, rhinoplasties were a common order of the day and practiced by the Koomas or tilemakers—one of the lowest castes in India. Some of their surgical skill was gained by practice on the bodies of dead animals, a defilement that could not be tolerated by any but the vilest of outcasts. Such was the lowly origin of plastic surgery! During this period and extending into several centuries of the Christian era, prisoners of war were often mutilated. In peace time, mutilation was practiced as a penalty for various offenses. Significantly, in one of the conquered cities of India, nasal mutilation became widespread to such a degree that its name was changed to signify "City of Cut Noses."

Nasal restorations were obtained by the use of pedunculated flaps from the cheeks and forehead with the employment of a certain "cement." Whether it was this certain "cement," the composition of which but little was written, or the results obtained by restoring mangled parts, the practice of featural restoration became a prominent part of the medical writing of that period. The results of these cosmetic operations were gratifying, much to the displeasure of those in authority inflicting the punishment. And so, in order to insure the penalty, the noses of criminals or enemies so mutilated were thrown into a fire with the belief that replacement would then be impossible. Rhinoplasty, then as now, was the most important phase of plastic surgery.

Celsus, a Latin physician who lived in the first century, has been called the Father of Plastic Surgery. A nobleman by birth, gifted with literary elegance and erudition, his influence and fame were such that he was admiringly known as the Roman Hippocrates. The scope of his writings covered an enormous field, in and out of medicine. His minute presentation of original featural corrections, instrumentation, together with the qualifications of a surgeon are prima facie evidence that he was a *medici* and not a *man of letters* as Pliny chose to designate him. The only remaining works of Celsus, *De Re Medicina,* contained, among the other data, authentic principles of plastic surgery. Published 1300 years after his death, this volume was one of the first medical books to be printed (1478), passed through more editions than any other scientific treatise of that era.

The distinguished physician during the second century in Rome was Galen, the most prolific of all ancient writers. His contributions were a gigantic encyclopedia of the knowledge of his time, together with countless original observations, including plastic surgery. A tireless worker, he wrote on practically every subject in the art of medicine, clearly explained and properly recorded—after the manner of a dictator—to his own satisfaction. In the latter respect, he was "allergic" to the talent of others and was a past master in the art of underestimation. He bathed in his own applause and was pedantic to the n'th degree. He was in his sixth heaven and rising rapidly when uncorking the vials of his venom on his adversaries. Aside from this pomposity—achievement must be respected. He was the most gifted and learned physician of his time and when he died, the markers on the road to progress were lost for centuries to come.

From the time of Galen to the Renaissance is known as the Middle Ages of Europe. It is a period of morbid fascination when meaningless incantations replaced medicine. Mysticism, spiritualism, superstitions and other varieties of magic were the order of the day. Miracles were related by everyone and the belief in them considered commonplace. Holy men and women each specialized in the cure of a specific illness, so that the number of saints became legion. It was an age which believed more of the irregular medicine without confirmatory evidence or scientific thinking. Translations of the ancients from Arabic to Latin to Greek and from Herbrew to Syriac to Egyptian—back and forth—were the fixed pattern of disseminating information, but not new ideas.

Surgery was considered beneath the dignity of the physician, ecclesiastical opposition strictly prohibited dissection and popular prejudices were antagonistic to all things medical.

Surgery fell into the hands of the so-called "barber-surgeons" who matched their technique with the tempo of their time. A calloused mind has no vulnerable spot and so these barber-surgeons not only flourished but became incorporated. And so we quietly leave the dark middle ages of Europe—when the surgeon was synonymous with a murderer—medicine became decadent and the Black Death accounted for sixty million victims.

Not until 1140 was issued the first European law of medical licensure. Those failing to pass the examination by the "board" were imprisoned and all their goods sold at auction. It seems rather strange that the above act preceded the establishment of the first medical school by a century—sort of putting the cart before the horse.

It was Frederick II who established the first medical school at Salerno about 1240. He was a fine linguist, excellent observer and a keen investigator. The requirements he proposed consisted of three years pre-medical, five years of medical curriculum, medical board, and, since there were no hospitals, the young medico must serve as assistant to an established physician for a full year. A short flight on the magic carpet of time—seven centuries later, the requirements are the same!

Had it not been for the lawyers of Bologna demanding an autopsy in cases of suspicious deaths, pigs might have continued to be used for anatomical studies. Thus, dissection was brought to the cadaver! In 1315, Mundinus, University of Bologna, obtained two female cadavers which he dissected for his students. A year later, as the result of these two dissections, he published a hand-book of anatomy which for the next 200 years was the accepted and accredited *Anatomia* of all medical schools.

Branca of Sicily, regarded as the first surgeon of the world, 1442, established a reputation of building up noses from the skin of the face. He bequeathed the art to his son, Antonius, who was even more skillful than his father. During the following century, the art was practiced in a near-by Italian province by Vianceo, and after his death by his son, Peter, and a nephew, Barnardin. It was then passed on to the Bojani family, where the art seems to have halted—but only temporarily.

To most of us, Leonardo da Vinci (1452-1519), is remembered for that inscrutable smile on the countenance of Mona Lisa del Gioconda and for the Last Supper. He was the most versatile of men and the greatest anatomist of his epoch. He was the first to dissect many bodies, and drew so accurately that these pictures are unsurpassed today. His notes and drawings, unknown to most of his contemporaries, have only recently been recovered, and includes everything from surface anatomy to osteology and the viscera. He was the first to include cross-sections, so vitally necessary for operative surgery. The anatomical drawings prior to da Vinci for any purpose—by comparison—appear as if they had been executed by a school child. It has since been written that the notes and anatomical drawings by da Vinci was the most precious legacy bequeathed to mankind since the Greeks.

Andreas Vesalius, 1514-1564, the most commanding figure in European medicine since the time of Galen, was made Professor of Anatomy at the University of Padua at the tender age of 23. As a student he was dissatisfied with the usual pig dissections carried on by the barber-surgeons or demonstrators while the students only observed. His anxiety for genuine knowledge was so great that he resorted to robbing graves for human dissection, and soon gained a reputation that was destined to place him among the immortals in the hall of medicine. He dissected as he lectured and made anatomy so interesting that his amphitheatre was always crowded. He was the first to make students dissect the human body and the first time in the history of the world—the "medical bible" was opened and studied by the embryo medico. His seven books, "Fabric of the Human Body," are a masterpiece of anatomical knowledge. Like all pioneers, he paid the price for such a radical publication. Truth has a penchant for dropping into that familiar pasture of jealous friends, whose razor-edged attacks grooved deeply into the victim's mind. Vesalius abruptly resigned, burned all his writings, and never dissected or taught again.

Gaspare Tagliacozzi, 1546-1599, was the first to describe minutely the rhinoplastic procedure which is now referred to as the "Italian Method." He constructed an entire nose by means of a pedicle flap from the arm and eliminated the facial scars from the previously employed cheek and forehead method. For thirty years he was Professor of Anatomy and Surgery at University of Bologna, the most ancient seat of learning in Europe with the possible exception of Salerno. The anatomical theatre of the University of Bologna was a showplace in more than one sense. In this famous amphi-

theatre lectured Maria Valsalva, Luigi Galvani, Marcello Malpighi and other luminaries whose fixed pattern of basic principles must be haloed in dignity. Of the female professors, Novella d'Andrea, who taught anatomy, is particularly remembered. Legend has it that her beauty was so staggering that she lectured veiled:

> Lest the Students
> Should let their eyes wander o'er her
> And quite forget their jurisprudence.

In other lecture halls of the University mused and dreamed men who were destined to become immortal: Boccaccio, Dante, Copernicus, Luther, Erasmus, Petrarch, Aristo, Tasso! A humorous story went the rounds of the Bolognese barrooms that Tagliacozzi made noses for his patients from the gluteal regions of other persons. His only book published on this subject, in 1597, covered 298 pages, illustrated with woodcuts, went through a second edition, and shortly before his death an edition appeared in Frankfort, Germany. More than 200 years later, in 1831, to be exact, this book was reprinted.

After the death of Tagliacozzi, the lay and particularly the ecclesiastical groups pronounced his work a sacrilege—for attempting to improve upon God's handicraft. The prevailing punishment was dealt to this brilliant surgeon. His body was exhumed and thrown out of holy ground—the cemetery. It is almost inconceivable that one whose name, bordering on idolatry, would be so desecrated. The road of the pioneer is always obstructed, but time is a great healer. Today an expiatory statue of Tagliacozzi, holding an artificial nose in his hand, adorns Bologna.

During the 17th century, surgery was excluded from the curriculum at the University of Paris and witchcraft wrote a bloody chapter for the history of the world. Surgery was still not only questionable but exceedingly dangerous and advanced no futher than Paré. Anatomists worked secretly and diligently, with the result that approximately half the structures of the body bear their names.

Facts discovered in the 17th century often produced theories in the 18th. In this era, Morgagni, last of the line of Italian genius at Padua, described the diseased body with such accuracy that he created the science of pathology. Surgery was making progress in France, considered a sociologic crime in Germany, and in England remembered as the "Golden Age of Quackery."

Modernization of medicine began in the 19th century with the extension of physics, chemistry and biology, all branches of medicine and surgery developed with rapid strides.

After the time of Tagliacozzi, there was a lapse of more than two centuries before the next volume on plastic surgery made its appearance. This was in 1818, when von Graefe published his "Rhinoplastik," which included his original operation for congenital cleft-palate.

The barber-surgeon crudely plied his trade in England until 1745, when an act declared, "That the business or trade of a barber is foreign to and independent of the practice of surgery." The Anatomy Act in 1832 legalized human dissection only after a most shocking chapter had been written by the grave robbers and professional murderers—particularly the "firm" of Burke and Hare.

The greatest anatomist of the 19th century was Jacob Henle, the grandson of a rabbi. His volumes covered a most comprehensive survey and the drawings were by Henle himself, executed with such rare skill that Darwin declared them to be "the best ever published."

The introduction of the hypodermic syringe by Pravaz about 1851, produced one of the most useful instruments in medicine and surgery; likewise, one so frequently abused!

The brilliant achievements of Louis Pasteur, Robert Koch and Lord Lister are of such historical character that volumes have been written about each. Suffice it to say, that the germ theory of disease and the rôle that each of these pioneer investigators played, has been hailed as one of the ten greatest discoveries in the history of the world.

Lister made surgery clean! Laudable pus with its "surgical stink," the filthy, bloody old operating coat and dirty instruments were forever banished. Only one more item remained to parade its magnificence and to complete the surgically set stage—anesthesia. Nitrous oxide (1844), ether (1846) and chloroform (1847), was America's gift to the world.

Although cocain was isolated from cocoa leaves by Albert Nieman in 1858, a quarter of a century passed before its practical application was recognized by Carl Koller in opththalmic surgery, at the suggestion of Sigmund Freud, of psychoanalytic fame. Within a year cocain was used all over the civilized world. About this time William Stewart Halstead did the first nerve (mandibular) block, and James L. Corning demonstrated infiltration and spinal anesthesia. The toxicity of cocain stimulated the production of a synthetic relative which would be less toxic, of greater therapeutic value and non-irritating locally. After much research novocain was produced by

Alfred Einhorn in 1904, and introduced by H. Braun of Germany the following year. Braun went a step further—he added suprarenin to allay absorption and check bleeding.

The material for plastic corrections were plentiful enough when one remembers the universality of syphilis, especially in Southern Europe, and the fact that belligerent gentlemen went about sword in hand, slitting and cutting off the noses of offensive rivals for the pleasure and satisfaction of their fair ladies. An individual with an unmutilated nose in France, Spain or Italy was indeed a rarity. Comparable to this, German university students were exceedingly proud of their facial scars. Since duels were common, Hoffacker of Heidelberg reported many cases of facial restorations. The manner in which these students exhibited their engagement scars bordered on a superlative vanity—the nearer the eye the greater the badge of honor!

The need for plastic surgery aroused the French and Germans as well as our own Thomas D. Mutter of Philadelphia, who achieved fame as a surgeon. As a recent graduate, Dr. Mutter wrote extensively on corrective surgery of the mouth and nose, deformities due to burns and cleft palate. About the same time (1842), Joseph Pancoast of Philadelphia and J. Mason Warren of Boston also published clinical reports of operations for the correction of nasal and facial deformities.

In 1845, Diffenbach published a book on plastic surgery. He developed the double-faced flap, realizing the importance of an inner lining for the nasal cavity. His description of a method for the building of a columella and the raising of a flattened nasal tip are truly remarkable. Langenbeck succeeded Dieffenbach and became the greatest clinical surgeon and teacher of his day. More than a score of original operations are credited to his name, including plastic surgery of the lip, palate and nose.

Among the many others associated with the development of plastic surgery are such names as Lisfranc, Von Mangold, Gillian, Koenig, Lexer, Correll, Reverdin, Thiersch and Wolfe. The latter three are responsible for their original methods of skin grafting. Ollier, 1861, was the first to employ a living bone transplant for the construction of a nasal bridge, and, in 1896, Israel transplanted a free bone graft taken from the anterior surface of the tibia for the reconstruction of a saddle nose. The utilization of rib cartilage, original with Charles Nelaton about 1900, also for the correction of a saddle deformity, has many followers. The same year the injection

of paraffin advocated by Gersuny was hailed as one of the century's greatest boons to mankind. His first case to correct a depressed nose was reported as a satisfactory cosmetic result. Gersuny did not stop there but employed paraffin anywhere about the body to restore the normal contour. This so-called "filling method" won acclaim and favor on account of its simplicity. Indeed, the author of a book on plastic surgery published in 1911, devotes 129 pages minutely describing the technique and extolling its virtues. However, this short-cut method to plastic surgery soon fell into disrepute on account of its disastrous results and of the grotesque deformities resulting. In retrospect, the injection of paraffin which, from its inception was spectacular, became controversial and within a decade was relegated to the scrap heap of modern plastic surgery.

Severe facial injuries during the early part of the World War were treated as follows: After primary healing had occurred, skillfully constructed prosthetic appliances were provided to cover the defects. These were attached to the face by means of spectacles. Although the transformation almost approached the normal in appearance, nevertheless the patients were never happy wearing what they termed "tin faces."

During the World War a conference of the Allied Powers created a separate department for the treatment of facial injuries and those requiring plastic operations. Attached to these specialty hospitals were nose and throat specialists, eye surgeons, brain surgeons, dental surgeons and others trained in head surgery by short intensive courses both here and abroad. All the heretofore known methods were employed and scrapped with great rapidity as improvements were devised. Their reconstructive work quickly began to bear fruit. Some of the most intricate and difficult problems were solved. The results were miraculous. A new difficult specialty was created in a few short years that otherwise would have taken a century to accomplish. These war pioneers, working under pressure, were true exponents of modern plastic surgery.

Major Harold D. Gillies of England deserves particular praise for his masterful advances in facial plastic surgery, during and after the World War. For his untiring efforts and brilliant results he was knighted by King George V.

The war ended. Plastic surgery continued for those facially maimed until restoration was complete. This formed an excellent basis for civil work. Those with irregular or distorted features came to the plastic surgeon, as well as those whose livelihood depended upon their appearance.

In 1915, Eitner reported a large number of saddle noses corrected with animal (elephant) ivory, and stated, "While implantations of cartilage always showed considerable contraction and while even pieces of bone were subjected to partial resorption, the ivory remained unchanged."

John B. Roberts of Philadelphia, John Staige Davis of Baltimore, Blair and Brown of St. Louis, Pierce and O'Connor of San Francisco, Brown of Milwaukee and the Mayos have added considerable to the many perplexing problems of this highly specialized field. Their works are of modern caliber with much original thought.

The late Jacques Joseph of Berlin may be reverently called the Father of Modern Plastic Surgery. No amount of praise or the finest shading of rhetoric can give adequate justice to this master surgeon. That he displayed positive genius is adequately confirmed by the great number of his original operations. Of these he is particularly acclaimed for correcting all nasal deformities by the intranasal route (with no external scarring) which consigned all such previous operations to obscurity. In addition thereto, he displayed his mechanical inventive genius by devising a complete set of instruments—a most necessary prerequisite—in order to employ this modern technique. He was an artist to his finger tips, with a perspicuous mind, a quality so rarely possessed. That he brought the art of plastic surgery into modern perfection may be attested by his extraordinary fecundity, coupled with the versatility of his writings. Physicians from all parts of the world studied under this master, and his technique is the only accepted one today. About ten years ago, he published his three volumes entitled "Nasenplastik und Sonstige Gesichtplastik nebst einem anhand über Mammalplastik und einige weitere operationen aus dem gebiete der äusseren Körperplastik—Ein Atlas und Lehrbuch von Prof. Dr. J. Joseph, Berlin, 1928." This text is written in an exceedingly clear style, profusely illustrated with exquisite drawings and photographs, so highly essential in any type of surgery. Destiny had placed an assignment upon Joseph, so well executed, that the more recent works are but a feeble attempt. True, advances and refinements have been noted in the past twenty years, but Joseph still remains as the greatest of all plastic surgeons.

And then the Nazi terror. The great Joseph fled to Czechoslovakia for safety. The savant's son-in-law, coming to his financial aid, was arrested while secretly attempting to cross the border. When the dire news reached Joseph, the shock proved fatal.

WIDENER BUILDING.

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XXXIX

VALUE OF THE VESTIBULAR TEST IN NEUROLOGICAL DIAGNOSIS*

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The vestibular tests may offer considerable aid in solving certain neurological problems in which intracranial localization is of prime importance. They may be corroboratory in character and serve to confirm data obtained by other studies. Then again, they may supply information which gives direction to otherwise apparently conflicting observations, and on occasions they may be the only finding to point clearly to a solution when all other examinations give vague, inconclusive or negative results. On the other hand, it must be realized that not a few cases are encountered, particularly of lesions in the supratentorial region, in which the vestibular manifestations are insufficient to justify a diagnosis of a definite localization, and yet a lesion is found at operation, having been localized by other means. Even under such circumstances, the vestibular examination is highly useful, since it aids in excluding subtentorial areas of the brain as possible sites of lesions.

In examining the vestibular apparatus the test is applied not only to the internal ear but also to a large portion of the central nervous system. By this form of examination the intactness of the internal ear, the eighth nerve, as well as that of the various afferent pathways and intracranial structures through which these fibers pass, is established. The tests place one in a position to state whether the lesion is peripheral or central, and, if central, where it is probably located. The progress of a lesion involving the vestibular pathways can also be frequently determined by repeated vestibular tests. Occasionally it is possible by these tests to localize the side on which the lesion is present when other methods fail.

In most instances the caloric test is employed instead of the turning test, inasmuch as the information obtained from the former is more accurate and reliable, since each semicircular canal is tested

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separately. It should be remembered that the tests are used mainly as an aid in the localization of a lesion and not to determine its nature. Moreover, conclusions or impressions as to localization should be reached independently of the neurological findings. It should also be borne in mind that the vestibular findings elicited by means of the caloric tests are influenced not only by the presence of the lesion itself, but also by the resultant intracranial pressure. Not without importance is the degree of co-operation received from the patient. Proficiency in the proper evaluation of these tests depends upon the experience of the examiner and can be acquired only by the study of many cases. Standardization of the technique and of the interpretation of the findings is essential. After careful notation of the vestibular findings, analysis should be based on the present-day knowledge of the vestibular pathways. The latter is based upon clinical experience, the result of many examinations, and correlative studies of neurologic, operative and postmortem findings.

The differential diagnosis of a peripheral and central lesion affecting the eighth nerve is based on the recognition of certain general principles and is of importance in localization by means of hearing and vestibular tests.

A peripheral lesion of the labyrinth or of the eighth nerve is suggested by the following findings: In a peripheral lesion all the responses to stimulation are impaired, and conversely the presence of any one normal response suggests a normal labyrinth and an intact peripheral eighth nerve. Spontaneous nystagmus in the vertical plane, either upward or downward, indicates a central lesion. If stimulation produces a "perverted" nystagmus, for example, an oblique or vertical instead of a horizontal nystagmus, then again the lesion is probably central.

If the findings suggest a central location of the lesion, then the next problem is to identify the seat of the lesion more definitely. The facility with which one is able to do this depends in a great measure upon how well one is able to visualize the various pathways constituting the vestibular apparatus. The simplest method of procedure is that of elimination; beginning with the labyrinth, proceeding centrally, one considers each structure separately:

1. If good hearing and one or more responses on stimulating the labyrinth are found to be normal, the labyrinth itself and eighth nerve may be considered uninvolved.

2. For information relative to the medulla, including the inferior cerebellar peduncle, the responses obtained on stimulating each horizonal canal separately are studied. The test is performed routinely by tilting the head back 60 degrees after douching. If this produces normal horizontal nystagmus and vertigo with normal past-pointing, these structures are probably not involved.

3. To determine the integrity of the pons, the responses obtained from the vertical canals are analyzed. They are tested with the head in the upright position. If this examination shows normal rotary nystagmus with vertigo, past-pointing and falling, it suggests uninvolved pathways in the pons and middle cerebellar peduncles on the side douched.

An evaluation of caloric tests in posterior fossa lesions based on an analysis of forty verified cases was carried out a few years ago in this hospital. Information of considerable value was obtained. Twenty-two of these cases were verified cerebellar neoplasms. A resume of the diagnoses, otological and neurological, and their comparison with the operative and postmortem findings showed:

Complete agreement in localization	11 cases
Disagreement	4 cases
Partial agreement	2 cases
Incomplete examination	2 cases
Difference as to side of localization	2 cases
Otological findings verified—patient not examined by neurologist	1 case

Four of these cases showed hearing impairment or total loss of hearing, of the nerve type, on the side ipsolateral with the lesion. One of these had total loss of hearing and vestibular function. The localization made by the otologists was an angle neoplasm. The neurological diagnosis, however, was a cerebellar lesion with involvement of the pons. A right cerebellar cyst was found at operation.

The findings on caloric stimulation, characteristic of a posterior fossa lesion with localization in the cerebellum, are:

Diminution or absence of vertigo.

Diminution or absence of past-pointing, or past-pointing in the wrong direction.

Normal nystagmus, unless pressure upon the brain stem causes deviation from the normal, such as spontaneous vertical or oblique nystagmus, or a perverted nystagmus on stimuation.

Normal hearing. Occasionally there may be present, from pressure upon the eighth nerve of a cerebellar neoplasm, hearing impairment of the nerve type.

Spontaneous past-pointing in one direction or, in the absence of spontaneous past-pointing, an inability to produce a reactive past-pointing, is suggestive of a lesion in the cerebellum. On the other hand, when we are able to produce normal past-pointing of each arm in either direction, it is probable that the cerebellum is intact. As in all methods of diagnosis, there are exceptions to this rule, but in general, the above holds true. Of the forty cases studied, eighteen were in the so-called extra-cerebellar group (pontine angle and pontine lesions).

The most common symptom in this group was loss of hearing; this occurred in sixteen of the seventeen angle tumors and in fourteen of these it was the earliest complaint; in two others it was preceded by tinnitus. In two, headache was the first symptom noted. Nine patients complained in addition to deafness, of vertigo, headaches and dimness of vision. Three had attacks of transient blindness. Eleven patients complained of disturbance of gait, weakness or clumsiness. Two complained of speech disturbance and difficulty in swallowing.

A diagnosis of an angle neoplasm when fully developed, based on vestibular and cochlear findings, presents little difficulty. In the earlier stages tinnitus and a partial impairment of hearing of the nerve type with vertigo, not uncommonly is diagnosed Ménière's disease. With further changes in the cochlear nerve, especially diminution, and later loss of vestibular function, the diagnosis becomes quite obvious. Since a fair percentage of all brain tumors are cerebello-pontine angle tumors, a method that is so helpful in either localizing them or excluding them from this region, immediately becomes of utmost importance.

While it is true that the neurologist can diagnose angle lesions without a vestibular examination, it is equally true that they must wait for the appearance of general clinical phenomena before such a diagnosis can be made. On the other hand, with a vestibular examination, a diagnosis can be made rather early, at a time when operation promises the best results. The number of conditions that could simulate the vestibular phenomena of a cerebello-pontine angle lesion

are very few, so that the vestibular tests furnish a most definite means of determining the presence or absence of such a lesion. The following phenomena are usually regarded as characteristic of the findings in cases of angle tumor:

1. On the side of the tumor, a loss or impairment of auditory and vestibular function.

2. On the opposite side, a normal auditory function, normal responses from the horizontal canal, but absence of responses for the vertical canals.

In brief, otologic examination shows a loss of function on both sides except in the cochlea and horizontal canal of the ear opposite the tumor.

In none of the five cases, the group mentioned above, which showed changes on stimulation of the opposite canals, was this finding observed. On other occasions I have been able to demonstrate this finding. Where such a combination of both peripheral and central phenomena exists in the same patient, the lesion is located in all probability in the cerebello-pontine angle. A tumor in this situation destroys the eighth nerve and produces a typical picture of a peripheral lesion on that side, and, in addition, pressure phenomena which manifest themselves by various so-called "central lesion findings" on testing the opposite canals.

The peripheral lesion element of this symptom complex is not difficult to determine, as the patient usually has a total loss of function of the eighth nerve in both branches, or if the hearing is partially retained, the responses from the horizontal and vertical canals on that side will be completely absent, making the evidence of peripheral lesion on that side fairly clear. It is the "central lesion" element of the picture that requires the greatest interpretive skill. None of our cases showed the typical findings on stimulating the opposite canals. There may be variations, as in five of our cases, from this characteristic finding, making the interpretation far more difficult. For instance, the patient's hearing on the unaffected side may not be normal, due to a long-standing catarrhal or suppurative process, or perhaps to an old pre-existing eighth nerve or cochlear degeneration. Again, the horizontal canal on that side may respond, but in such a manner as to make it difficult to determine whether or not such a response is impaired as compared to the fully normal response. Cases of this type often exhibit other phenomena indicative of a central lesion, such as a vertical nystagmus, up or down or per-

verted nystagmus on the unaffected side, or a definite disproportion between eye responses and vertigo. One of our cases showed the presence of a perverted nystagmus on stimulation of the opposite horizontal canal, i.e., vertical nystagmus upwards and downwards following such stimulation. In three there was observed interference with or wrong past-pointing, with either hand on stimulating the opposite canals. Twelve of the cases of angle tumor gave normal responses on stimulating the opposite vertical canals. Careful observation makes it possible to detect, in some instances, a definite difference in the activity of the horizontal as compared with the vertical canals.

An isolated loss or marked diminution of responses from the vertical canals on the opposite side, the occurrence of perverted nystagmus on stimulating the opposite horizontal canal, and abnormalities of past-pointing—all of these phenomena appear to be caused by pressure of the tumor upon the brain stem. In a well advanced growth in the angle, abnormalities in past-pointing are frequently observed either in the form of spontaneous past-pointing or an absence or impairment of past-pointing after ear stimulation. Crossed past-pointing was not observed in our cases but has been noted on other occasions—both arms past-point inward or outward.

In addition to those lesions already discussed, the diagnosis of the following lesions may be further clarified by means of vestibular tests:

In supratentorial neoplasms there is often an increased susceptibility to stimulation, resulting in exaggerated nystagmus with a corresponding increase in vertigo and past-pointing. Conjugate deviation of the eyes on vestibular stimulation may suggest the presence of a lesion on the side toward which there is deviation.

Frontal lobe neoplasms with cerebellar signs may occasionally be identified correctly by excluding, by means of the vestibular tests, the posterior fossa as the site of the tumor.

Ménière's disease: The vestibular-cochlear findings in this condition in the early stages show no departure from the normal. Repeated vertiginous seizures result in increasing hearing impairment of the nerve type. The hearing impairment in severe cases or those of long duration may be marked, and in some instances almost entirely gone. The vestibular responses show either no changes from the normal, or, if the condition has lasted for some time, moderate diminution of all responses.

CONCLUSIONS

1. Vestibular tests are of considerable assistance in the diagnosis of brain stem lesions and particularly in the localization of expanding intracranial lesions. They are useful:

In the differential diagnosis of peripheral and central lesions of the eighth nerve;

In the differential diagnosis of an infra- from a supratentorial lesion;

For more definite location of posterior fossa lesions: cerebellum, pons, cerebellar peduncles, pontine angle, etc.;

In the early diagnosis of cerebello-pontine angle neoplasms;

As aids in localization by exclusion;

For the evaluation of Ménière's syndrome.

2. To be of value these examinations should be done by none other than those thoroughly trained in the technique and the interpretation of the tests.

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Clinical Notes

XL

TRANSVERSE FRACTURE OF THE PETROUS PYRAMID

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Fractures which involve the petrous pyramid are classified as longitudinal or transverse. Of the two, the longitudinal is by far the most frequent. The longitudinal fracture never involves the labyrinthine capsule directly; the transverse variety always damages the capsule. The longitudinal is a middle fossa fracture; the transverse, usually due to force applied to the occipital or occipito-mastoid region, is, for the most part, a posterior fossa fracture. Transverse fractures are subject to considerable variation in position and direction. There are two extreme types, the internal, which courses through the internal auditory meatus and shatters the cochlea in its antero-internal portion, and the external, which passes through the entire internal ear, cochlea, vestibule and fallopian canal. In about one-half of the cases the middle ear is spared entirely, but when injured the damage affects only its medial wall (eustachian tube, knee of the facial and the foot plate of the stapes). Fissures between the windows are frequently found; the promontory may be torn loose; the foot plate of the stapes may be luxated.

Such a labyrinthine fracture may cause a hemato-tympanum or an escape of cerebrospinal fluid into the tympanic cavity by injuring the mesial wall of the middle ear, but it rarely, if ever, causes a rupture of the drum and bleeding from the external canal. When bleeding from the ear is associated with total loss of function we are dealing with a combined transverse and longitudinal fracture. As it courses through the labyrinthine capsule the transverse fracture usually causes complete loss of cochlear and vestibular function, although this is not always true. Retention of one or the other function has been demonstrated in well authenticated cases.

The transverse fracture is essentially a fracture of the labyrinth. It may be macroscopic or microscopic. If macroscopic, it is visible

at operation and at autopsy. The microscopic fracture is invisible at autopsy but can usually be detected on the roentgenogram and always histologically. It may take the same course as the transverse fracture but it may also remain strictly intracapsular.

The radiological demonstration of the longitudinal fracture offers many difficulties even when attempted early, and after the lapse of a month or two becomes practically impossible. The transverse fracture can usually be delineated by x-ray, often even when microscopic, and this is true for many years after the injury. This is due to the fact that the capsular fracture does not close by bony union or does so very imperfectly. The reason for this peculiar phenomenon is found in the histological and biological characteristics of the bony labyrinthine capsule. The capsule is composed of three distinct layers or types of bone: (a) an endosteal or internal layer, (b) a periosteal or external layer, and (c) the enchondral layer which is the thickest of the three. This enchondral layer is primary bone, and never becomes adult bone and shows little or no tendency to regenerate. It is particularly abundant in the region about the windows, in the deeper portions of the internal auditory meatus and about the first and second turns of the cochlea. This incapacity to regenerate is important for two reasons. It permits the radiological delineation of fractures of the labyrinthine capsule many years after they occur. When the line of fracture puts the internal ear into communication with the middle ear, the danger of a late meningitis is always present. Such meningitides have been reported as late as sixteen years after the fracture occurred.

REPORT OF A CASE

CASE 1.—Margaret M., age 13, who lived in a neighboring city, was injured in June, 1930, when she was knocked off of a bicycle to the pavement by a passing automobile. She presented herself for examination on October 8, 1930. The following history was obtained from her mother and her family physician. She was unconscious for about four hours. The accident occurred at 6 p. m. and her doctor was able to communicate with her the following morning. At this time she was able to hear him. By evening that same day she heard with great difficulty, and during the course of the following month she lost her hearing completely. X-rays taken at that time did not reveal any skull fracture. There was no bleeding from the nose, mouth or ears, but she vomited blood for 24 hours and at intervals thereafter. The girl had vertigo on the morning following the accident, but her doctor did not consider this as violent in character.

This was present even in the recumbent position but was aggravated by sitting up.

Past History: Scarlet fever one year preceding but with no sequellae referable to ears or hearing. No previous history of otitis media.

Present Complaint: Total deafness, vertigo in attacks when making sudden movements, tinnitus and occasional headaches.

Examination: No facial paralysis. Pupils equal and react promptly. No ocular evidence of increased intracranial pressure. Vision normal. No evidence of fracture in either external canal. Both drums relatively negative; no scars, no perforations. A loud shout could be appreciated by the right ear but not interpreted (left ear masked by Bárány noise apparatus). Left ear totally deaf. There was a spontaneous rotary-horizontal nystagmus to each side in position I, head erect. There was a spontaneous past pointing to the right with the left arm. The Romberg was negative. In forward and backward gait with eyes closed she deviated first to one side and then to the other. Turning tests: 10 turns to the right in 20 seconds produced no nystagmus, neither did 10 turns to the left; 10 turns to the right in 10 seconds produced no past pointing with the right arm but a slight past pointing to the right with the left arm. This corresponded to the spontaneous past pointing previously noted. Ten turns to the left produced no past pointing with either arm. Five turns to the right produced no falling reaction, nor did five turns to the left. The caloric irritability was not tested at this time.

Impression: From the results of this examination I believed that we were dealing with a bilateral transverse pyramid fracture.

On October 22, 1938, I re-examined this patient, now 21 years of age. She could not hear speech but said that she could hear music. She had learned to read lips and had completed a course as a beauty operator. The hearing for the voice was as before. An audiometric examination of the right ear, while the left was excluded by a Bárány noise apparatus, showed a subtotal loss of hearing. The left vestibular apparatus could not be stimulated with any amount of ice water. The right did not react until 90 cc. of ice water had been used and then only gave a minimal response. X-rays in Stenver's position showed an irregular fracture line across the left petrous pyramid near its base, but no fracture could be demonstrated on the right side.

Comment: This proves to be a case of transverse fracture of the left petrous pyramid with a total left cochleo-vestibular paralysis and almost total loss of cochleo-vestibular function on the right side. The loss of function on the right side was probably due to massive hemorrhage into the right labyrinth, although nerve injury at the internal auditory meatus cannot be excluded. The case illustrates the fact that the petrous fracture was demonstrable by x-ray eight and one-half years after it occurred.

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XLI

POLYP OF THE TONSIL: REPORT OF A CASE

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AND

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Benign tumors of the tonsil are so rarely encountered that it seems worthwhile to record the following case:

REPORT OF A CASE

The patient, a girl, aged thirteen years, was referred by her family physician in July, 1933, for diagnosis and treatment of a tumor of the throat, which the child's mother had discovered accidentally about two weeks previously. The patient had no pain or soreness in the throat, nor complaints of any kind, and appeared to be in excellent health. There was, of course, no way to determine how long the growth had been present.

Examination of the throat revealed an encapsulated pedunculated growth, about five centimeters long and about two centimeters wide. The tumor was attached to the right tonsil, near the upper pole. A few lymph nodes were palpable in the right cervical region.

Tonsillectomy was advised, and at operation the growth was found to be attached to the upper pole of the right tonsil. The tumor mass appeared as an outgrowth from the tonsillar surface, near the margin (Fig. 1). It was pedunculated, with a smooth surface, and of a pale yellowish-gray color, mottled with red and purple. The growth was firm, but pliable, and its tissues were resistant to cutting and appeared to be fibrous. The growth measured 4x2x1.2 centimeters.

Microscopic examination of a section of the tumor showed that it was composed largely of connective tissue stroma which was well vascularized and covered by stratified squamous epithelium, which was normal in structure and arrangement. The vascular spaces throughout the tumor were numerous and large. There was considerable round-cell infiltration, and some lymphoid nodule-like structures were scattered throughout the specimen.

The pathologic diagnosis was benign polyp of the tonsil. Even though there was no evidence of malignancy, a short course of roentgen irradiations was administered to the cervical region. A month after the tonsillectomy the throat was completely healed and entirely normal. A period of more than five years has elapsed since the removal of the tumor, during which the patient has been well.

According to New and Childrey,[1] who have studied a large series of cases, a benign tumor of the tonsil or nasopharynx may be

Fig. 1. Photograph of gross specimen, showing the polyp attached to the upper pole of the tonsil.

papilloma, adenoma, lipoma, fibroma, myxoma, varices, angioma, chondroma, osteoma, polyp, teratoma, cyst or lymphoid tissue tumor. Some authors also classify mixed tumors as benign, but New and Childrey believe that these are of a low grade malignancy, and so list them separately. In their series of cases, studied at the Mayo Clinic from 1917 to 1930, forty-one tumors were situated on the tonsil or faucial pillars.

Among these were listed only two polyps, one of which later proved to be a malignant dermoid. The other was a true fibromyxomatous polyp in a man 44 years of age, which arose in the posterior wall of the pharynx. Of the papillomata, fourteen were in the faucial pillars, and ten on the tonsil. There was one case of lipoma in the tonsillar fossa. There was one fibrolipoma in the orifice behind the posterior pillar of the tonsil. One chondroma was observed in a woman, 30 years of age, and the patient was well three years after removal of the tumor. There were three cases of retention cysts in the pallatine tonsils, and three cases of lymphoid tissue tumor, two of which were pedunculated, in the tonsil.

Other cases of benign tumors of the tonsil have been described in the American literature during the last decade by Hara, Rosenberg, Smith and Frank.

Hara[2] reported a case of fibroma of the tonsil in a Japanese girl, aged seven and one-half years. The enlarged tonsil, which had caused no symptoms, was discovered by the school nurse. When it was removed, it was found that the growth originated from the fibrous layers of the tonsillar capsule and not from the pillars. The tumor was 50 millimeters in length and 20 millimeters in diameter at the widest portion, and was covered by a thin, nonvascular mucous membrane of stratified epithelium, beneath which was an indurated white connective tissue with some hyaline degeneration in the center.

The case reported by Rosenberg[3] was a dermoid cyst of the right tonsil in a man, aged 27 years. The growth had caused difficulty in breathing through the nose, coughing, difficulty in swallowing and occasional vomiting. The tumor measured 35 by 25 centimeters and weighed 7.3 grams. The surface of the growth was smooth, except for one area from which hairs were protruding. Microscopic examination showed numerous mucous, hair and sebaceous glands; the epithelium was thickened and of the squamous type.

Frank[5] reported three cases of papilloma, seen at Michael Reese Hospital in a period of six years, and Smith[4] noted another case of this type in his analysis of the pathologic material from 2,200 tonsillectomies at the Massachusetts Eye and Ear Infirmary.

The precise number of cases of benign tumor of the tonsil that have been reported in the literature is difficult, if not impossible, to determine. There is a wide discrepancy in the estimates of the number of cases of various types to be found in different reviews. For example, Liveriero,[6] in 1933, found reports of only 150 cases of benign tumor of the tonsil in the world literature. Of these, he stated that about one-half were fibromas, either pure or mixed. Hara, in the same year, listed only twenty-six cases of fibroma, to which he added one of his own.

The reasons for these discrepancies are not hard to find. Many of the reports are in otolaryngological journals which, among the foreign language periodicals, are not so readily available in medical libraries as the more general medical publications. Another difficulty in studying these cases is that they are not clearly segregated in the index. Some of them are reported in a series of cases of tumor, most

of which are malignant. In other instances, tumors of the tonsil are grouped with those of the pharynx and nasopharynx.

Finally, in studying the reports in the literature, it is evident that considerable disparity and confusion exist in the classification of various types of benign tumors of the tonsil. In order to increase knowledge of these lesions, it is important, therefore, that every case should be described carefully and in detail, so that it can be properly classified and evaluated by later students of the subject.

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XLII.

THE MECHANISM OF TRISMUS AND ITS OCCURRENCE IN MANDIBULAR JOINT DYSFUNCTION*

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ST. LOUIS

Trismus is the tonic contraction of the muscles of mastication, and varies in effect from a subpainful tenseness of movement of the lower jaw, due to mild reflex irritations about the jaws, to the extreme spastic closure seen in hysteria, tetanus, tetany, parotid inflammation, deep pharyngeal abscess, periostitis and fractures of the mandible. It may also be a symptom of organic disease, irritation occurring about the motor nucleus of the Vth nerve. In the marked painful types, the jaws are fixed together with little if any opening, by a strong continuous spasm of the internal pterygoid, the masseter, and the temporalis muscles. Not always an independent phenomenon, it is frequently associated with general convulsions.

Various degrees of trismus have been observed and selected from the cases being studied for neuralgia and ear symptoms originating in dysfunction of the temporo-mandibular joint.[1] The relation of trismus to various reflex dental irritations has been a matter of common knowledge. In numerous of these examples of the milder forms, trismus was relaxed by simply adding support within the intramolar spaces, extending the condyle ever so little from its impacted position. This illustrated the action of condyle movement in producing or accentuating the reflex effects. Unilateral reflex irritations producing trismus contraction of the masticator muscle group on one side only, with incoordinate action of the chewing muscles, are constantly seen in unbalanced occlusion, and constitute major dental problems.

The following series of twenty-three cases of trismus is classified to illustrate the mechanism by which sensory irritation is reflected from various districts supplied by the third division of the Vth nerve to its motor branches supplying the muscles of mastication.

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REPORT OF CASES

Group I: Originating in inflammatory irritations about the ear and jaws:

CASE 1. (190.) Miss C. Q., age 27, July 7, 1937. (Aural pain, right side; trismus.)

For two weeks patient felt "tightness, or slipping" deeply in front of right ear, which became a painful sensation when she attempted to open the mouth or close the jaws firmly. No illness or injury preceded the onset.

Positive findings: The mouth opened with incisors a pencil-thickness apart, with pain referred to right ear; tonsils small, chronic infected; teeth irregular, occlusion poor, right upper second molar carious, gum ulcerated; right mandibular joint very tender to palpation.

Course: Carious tooth extracted, baking of jaws by infra-red heat; trismus entirely relaxed in two days, pain relieved.

CASE 2. (196.) Miss G. B., age 65, August 2, 1937. (Soreness in right ear canal, thin discharge; mild trismus.)

Painful soreness in right canal two years, treated as furuncles at first, seemed to improve, the past four months entire lobe became inflamed, the canal closing and resisting all treatment.

Positive findings: Right ear canal closed with waxy, granular tissue, covered with thin serosanguinous discharge, the lobe swollen and excoriated; movement of lower jaw guarded, producing much pain in the right ear; occlusion poor, from various missing teeth, excursion of the jaw uneven, the condyles snapping backward on closure; biopsy of the right external canal showed epithelioma.

Course: Surgical removal of external canal and wide zone of periosteum, replacing the auricle; radium application on the fifth postoperative day; much improved, but some soreness persisted in right ear; trismus in short attacks when the soreness increased; recurrence of the epithelioma after 18 months, death April 3, 1939.

CASE 3. (259.) Mrs. J. W. G., age 29, June 6, 1938. (Aural pain, left, with swelling; trismus.)

For about one month had deep pain in left ear, and recently swelling of left canal with slowly increasing difficulty in opening the mouth, very little movement of the lower jaw possible the previous two days.

Positive findings: A large furuncle filled the left ear canal, with marked swelling over the adjacent parotid and mastoid regions; firm trismus prevented any opening of lower jaw.

Course: The furuncle was incised under gas, hot moist fomentations used continuously; relaxation of trismus and relief of the ear pain was complete in twenty-four hours.

Case 4. (265.) Mr. L. S., age 35, December 2, 1938. (Aural pain, right; trismus.)

Two weeks before examination, while chewing at mealtime, the patient felt a deep pain within the right ear canal, with sudden limitation of the opening movement of lower jaw.

Positive findings: Movement of the lower jaw was guarded, limited to one-half inch opening between incisors, and produced great pain in the right ear, as did firm chewing. Resting of jaws upon 2 mm. cork within molars on either side for test periods failed to relieve pain or trismus. X-ray of mandibular joints showed normal relations; x-ray of teeth showed large root abscess on right lower second bicuspid.

Course: Extraction of the abscessed tooth relieved the ear pain promptly, and the trismus relaxed in one week.

Group II. Originating in trauma about the temporo-mandibular joints:

Case 5. (148.) Mr. J. B., age 37, September 26, 1936. (Fracture of left arm, general contusions, trismus.)

Patient sustained accidental injury one week before with fracture of left arm and various contusions about the body. During this time had been unable to open the jaws except fractions of an inch at times, painfully.

Positive findings: In addition to splinting of the left arm for fracture, the patient assumed a flexed, half-sitting position in bed because of his general contusions. Complete trismus fixes the jaws in a closed position and forceful opening of the lower jaw was painful.

Course: As a test, cork discs of 4 mm. thickness were interposed between the posterior molars on each side. After fifteen minutes much of the jaw muscle spasm was relaxed, the patient voluntarily opening the jaws one inch without pain. The discs were carried

within the jaws a few hours every day, baking the district with infra-red heat at intervals. The trismus effect disappeared in three days.

CASE 6. (215.) Mrs. E. C. W., age 46, October 30, 1937. (Bilateral aural pain radiating to neck and throat; trismus.)

Positive findings: Dental plates were changed one week before, replacing poorly fitting ones used for four years. Trismus permitted opening of mouth one-half to one inch. X-rays of mandibular joints show only loosening and asymmetry of joint spacing.

Course: Light elastic headgear worn three to six hours daily to fix the position of the lower jaw. Trismus gradually relaxed in ten days with relief of all pain symptoms.

CASE 7. (262.) Miss R. M., age 31, November 16, 1938. (Aural pain and crackling noise right side; trismus.)

One week ago patient yawned, shoved her jaw to the left, with extreme pain and a snapping noise in right ear.

Positive findings: Opening of jaws was limited to 1 cm. in the incisor district and at the limit of opening the condyles shift to the left with a grinding noise in the right ear. Both mandibular joints extremely tender to palpation. X-rays showed joint space extremely narrow on right and excursion of condyles limited. Occlusion apparently perfect.

Course: Carried 2 mm. cork discs within right jaw six hours daily, baking with infra-red heat at intervals. Trismus and ear pain relieved in one week. To have upper impacted unerupted third molars extracted, and after a test period, occlusion balanced if necessary.

CASE 8. (266.) Mrs. F. E., age 22, December 19, 1938. (Aural pain, right; trismus.)

While eating one week before, suddenly had pain in right ear and spasm of jaw muscles, preventing occlusion of her teeth on right side—chewed with difficulty on left.

Positive findings: Opened lower jaw manually to one-half inch with much pain. Mandibular joints tender to pressure. X-rays (Fig. 1) show asymmetry of joint spaces, condyle excursion limited forward. Unerupted impacted right lower third molar.

Course: 4 mm. cork carried within right jaws, and 2 mm. cork within left two days with complete relief of symptoms. To have extraction of impacted molar.

CASE 9. (282.) Mrs. G. T. L., age 57, March 7, 1939. (Recurrent trismus of mild grade several years, continuous severe trismus five weeks, left aural pain, frequent blinding headaches.)

Five weeks before, several molars were extracted under gas, since which time patient could scarcely open the mouth at all. There was marked pain in the left ear when lower jaw was forcibly opened the small fraction possible. Because of other menopause symptoms, a course of theelin injections had been given.

Positive findings: The lower jaw was rigidly fixed, opening only 4 mm. between the incisors. Manual pressure upon the molar gum spaces enabled opening of the incisors 14 mm., with intense pain in the left ear. Palpation of the mandibular joints showed no tenderness, but the internal pterygoid muscles were tense and firm. No molar teeth present. X-rays (Fig. 2) showed condyles firmly impacted, and very little excursion on opening lower jaw.

Course: Dental wax skids were fixed within the molar spaces with great difficulty, and elastic chin strap applied for leverage. All painful trismus was relaxed by the following day, the temporary denture being worn five days with remarkable improvement, relaxation of lower jaw and relief of headache. At this stage the lower dentures were replaced, extending the vertical dimension 2 mm. more.

Group III. From abnormal movement of condyles, the result of various forms of malocclusion of original teeth:

CASE 10. (155.) Mrs. R. H. W., age 34, October 6, 1936. (Bilateral aural pain, worse right side; recurrent trismus.)

Attacks of pain about the ears for years, with grating noises, associated trismus, opening of the jaws varying from one-fourth to three-fourths of an inch during the attacks.

Positive findings: Excursion of lower jaw irregular, with fixation of the joints. The incisor opening varied within above fractions of an inch. Badly aligned natural teeth occluded poorly (Fig. 3).

Course: Balancing of occlusion was done at first with temporary splints which were worn six months before permanent reconstruction. Trismus and pain was entirely relieved during the use of the treatment dentures.

CASE 11. (212.) Miss J. C., age 30. October 19, 1937. (Aural pain left side, extending to neck and left side of the pharynx; trismus; dizziness.)

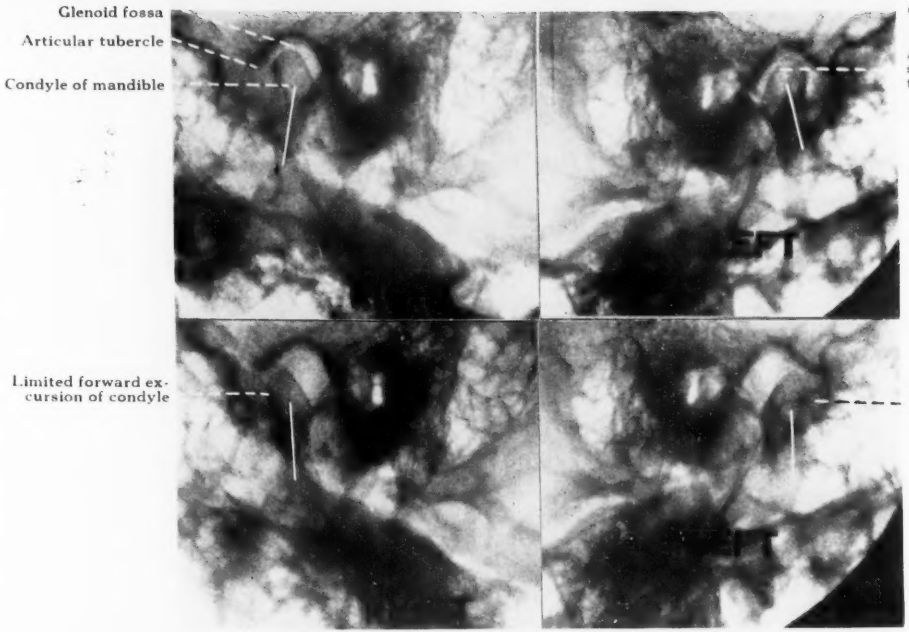

Fig. 1.—Case 8, Group II: demonstrates the production of mild trismus by an uneven jaw motion while eating. The tendency to produce this effect probably depended upon an unerupted impacted lower molar. The immediate attack was relaxed in two days by fixing the lower jaws against support between the molar teeth, splinted by the elastic chin strap. The presence of the unerupted molar places this case also in Group IV.

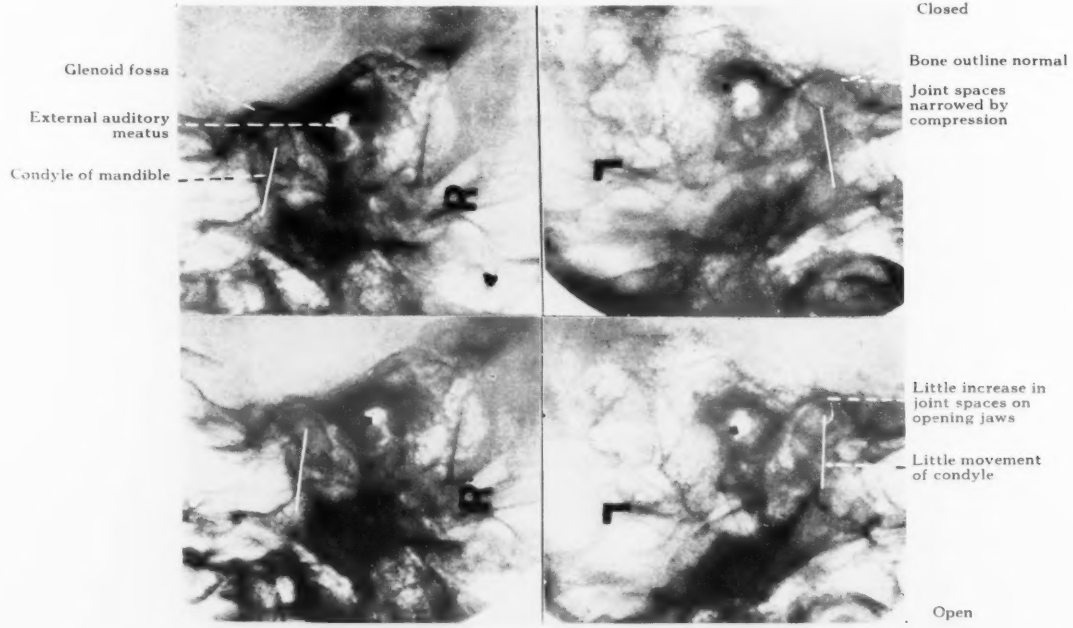

Fig. 2.—Case 9, Group II. The striking picture of continuous severe trismus remaining for five weeks, long after the mandibular joints were presumably injured by stretching under anesthesia. No reason could be found for the history of recurrent mild trismus unless it may have been from the inflammatory reaction about her carious teeth. From the above position relaxation of the trismus was accomplished solely by removal of the condyles from this impacted position, using dentures as a fulcrum and an elastic chin strap.

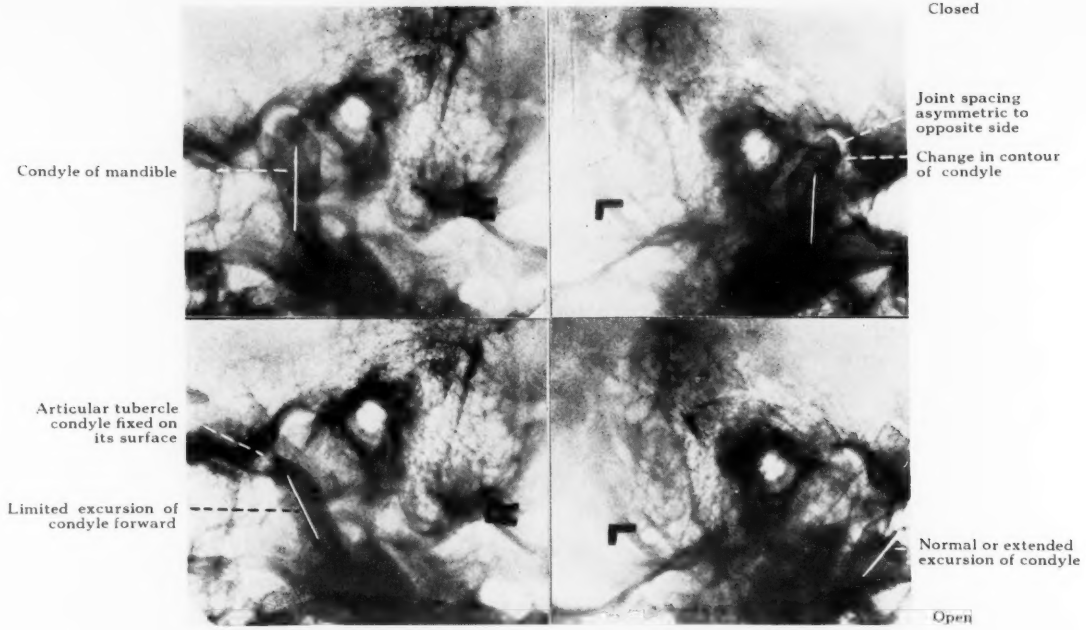
Closed
Joint spacing asymmetric to opposite side
Change in contour of condyle
Condyle of mandible
Articular tubercle condyle fixed on its surface
Limited excursion of condyle forward
Normal or extended excursion of condyle
Open

Fig. 3.—Case 10, Group III: shows the vicious circle established by malocclusion, producing destructive effects in the right mandibular joint; irritation and unilateral trismus (right), with fixation of the right condyle on the articular tubercle, when opened; closure proceeds with unbalanced muscle action and the malocclusing teeth further complicate the condyle movement on complete closure. Similar examples were reported by Steinhardt,[2] who correlated pathologic changes in the tubercle and condyle with x-ray findings.

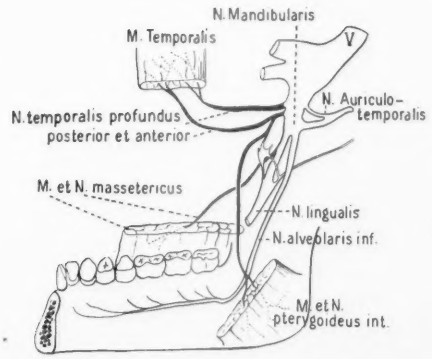

Fig. 4.—Schema showing branches of mandibular nerve, the motor or masticator nerves shown in black and sensory branches in white. Sensory irritation from all structures in this district, as from inflammation of the parotid gland, ramus of the jaw or pharyngeal tissues, produces trismus by contraction of the powerful masticator muscles. Relaxation of various grades of trismus in these studies by simple movement of the condyles away from the auriculo-temporal nerve and its sensory branches suggests that irritation by the impacted or abnormally impinged condyle is the mechanism of continuing the trismus.

Constant dull pain deeply in front of left ear for one year, began mornings and increased during the day, not affected by chewing. The pain radiated to the left side of the neck and into the left pharynx with a sensation of ordinary sore throat. Complete general study was made, including x-ray of cervical spine, all negative.

Positive findings: Opening of mouth limited to one inch, and very slight crepitation at the end of closure of the jaws. Dentition regular, with apparently good occlusion. Mandibular joints moderately tender to palpation. X-ray showed a peculiar enlargement of the joint space on opening with no movement of the condyles along the tubercle, no bone change, except a very slight flattening of anterior face of the left condyle.

Course: Patient carried 2 mm. thickness of cork within each jaw several hours a day for one week, with relief of pain and trismus and complete disappearance of dizziness. On this basis the dentist proceeded to balance the occlusion by the use of very thin gold overlays on the natural teeth, with continued complete relief of symptoms. Nine months later the trismus returned suddenly at the onset of acute pharyngitis, relieved after hot gargle two days.

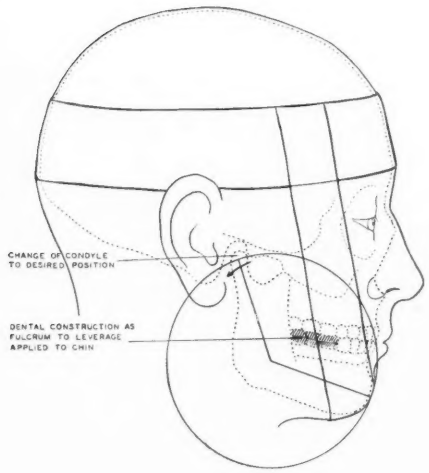

Fig. 5.—The use of elastic pressure applied to the chin as a means of increasing the speed of change of condyle position was suggested by Dr. Bland Pippin, Chief of Clinic, Washington University School of Dentistry. Both during the test periods, and during treatment, such use of the elastic seemed to "splint" the lower jaw, partially fixing it until painful effects within the joint subsided. It also seemed to control the subconscious effort of the subject to manipulate the lower jaw away from the newly interposed dental construction.

CASE 12. (221.) Miss R. G., age 27, December 11, 1937. (Aural pain left; trismus.)

Opening of lower jaw for past seven months restricted to about three-fourths of an inch. Variety of pains in the left ear on motion of the jaw.

Positive findings: Occlusion poor, all molars missing except the upper right, the upper alveolar ridge showing the "rocking chair" deformity. Opening of jaw restricted and the motion painful. Left mandibular joint extremely tender to palpation.

Course: Resting the left molar spaces upon a cork pack for ten minutes entirely relaxed the trismus and patient opened the mouth with ease to two inches. Patient was followed for one year by phone but refused to proceed with dentures.

CASE 13. (223.) Miss L. J., age 50, January 7, 1938. (Malocclusion; trismus; aural pain right.)

Right lower molars extracted one year before to relieve pain in the right ear. It increased rapidly with limitation of jaw motion.

Positive findings: Extremely bad occlusion of all teeth with absence of right lower molars. Trismus limits the mouth opening to one-half inch. Right mandibular joint extremely tender. X-rays show no bone change, joint spacing good, but right condyle does not move forward on the tubercle as far as the left.

Course: Wide opening of the mouth accomplished after cork pack support was held in right molar spaces one-half hour. Patient cannot be traced, but on last contact no dental work had been done.

CASE 14. (251.) Mrs. P. H., age 31, September 26, 1938. (Malocclusion; trismus; aural pain right.)

Beginning suddenly four months ago she began to have pain deep in the right ear on movement of the jaw, with the onset of completely fixed trismus two months later while chewing a sandwich. The severe attack lasted several hours and since then can open her mouth only fractions of an inch.

Positive findings: Smooth opening of the lower jaw to one-half inch with crepitation of right mandibular joint which is quite tender. Lower molars were missing on the right, the remaining teeth being in very poor occlusion. X-ray showed no bone change within the joints and excursion of condyles forward did not reach crest of tubercle; face of right condyle was flattened.

Course: Patient found greatest comfort with complete relief of all symptoms from carrying 4 mm. cork within the right and 2 mm. cork within the left jaws. Full mouth reconstruction and occlusion balanced, using denture on the lower right. No further symptoms.

Group IV. Originating in irritation from impacted molars:

CASE 15. (191.) Miss J. K., age 18, July 7, 1937. (Crepitus left ear; trismus; impacted third molars, left, upper and lower.)

About one year ago began to have stiffness in the jaw muscles, permitting the mouth to open about one inch, painful only when forced. Grinding noise in the left ear with each jaw movement.

Positive findings: Jaw movement is smooth except for crepitus in the left mandibular joint and opening of the jaw limited to vari-

ous fractions of an inch. X-rays showed beginning destruction of the left condyle and fixation of its excursion before the crest of the tubercle was reached; also impacted unerupted third molars on this side.

Course: Resting 2 mm. cork between the molar teeth relieved the trismus within 20 minutes. She was sent for extraction of the impacted molars, which was done. She has been observed one year and no return of symptoms; dental work deferred until necessary. (X-rays of mother's mandibular joints showed exactly the same picture and she gave a similar history, all of her teeth being extracted at the age of twenty for what was considered rheumatism.)

CASE 16. (225.) Miss A. A., age 32, January 21, 1938. (Aural pain right side; lower impacted third molars; trismus.)

Three years ago she began to have pain about the right ear which was worse toward the end of day and growing more severe. Opening of mouth seemed to be limited in proportion to intensity of pain.

Positive findings: Trismus limited mouth opening to one-half inch, the lower jaw snapping forward at end of opening and the condyles backward and to the left on closure. The right mandibular joint was extremely tender. (X-rays showed extreme looseness of the mandibular joint structures, with the left condyle resting unevenly against the tympanic plate; the right condyle assumed normal spacing within the fossa.)

Course: Trismus was remarkably relieved by resting 2 mm. cork within the posterior molars for a few minutes. The impacted lower third molars were removed with relief of pain and trismus. Full mouth reconstruction advised.

CASE 17. (238.) Mrs. G. A., age 39, April 22, 1938. (Trismus; impacted third molar, left; aural pain, left.)

Grinding noise in the left ear for four months and left aural pain recently, which is increased by closing the jaw.

Positive findings: Condyles make a grating noise and shift to right on closure. X-rays showed unerupted impacted lower left third molars and narrowing of mandibular joint spaces on the left side.

Course: Resting 2 mm. cork within the left jaw entirely relieved the usual pain on closure and mouth may be opened widely. Sent for removal of the impacted molar and dental reconstruction was advised.

CASE 18. (247.) Miss A. E., age 25, November 17, 1938. (Aural pain, right; recurrent trismus; unerupted impacted second and third molars, lower right.)

For eight years patient has had severe pain in right temple radiating to right ear; cold packs relieved it. For three years the right jaw joint had snapped on closure. The pain was increased when patient became hot with exercise.

Positive findings: On opening, lower jaw snaps at half-way point and at about the same distance on closure—while testing, jaw assumes complete trismus which lasted a few minutes. Both mandibular joints extremely tender, more marked right. X-rays showed flattening of face of right condyle and angulation of right glenoid fossa. Both second and third molars, lower right, have not erupted and showed impaction in the x-rays.

Course: Physician had already used 2 mm. cork within the right side for two weeks, greatly increasing the pain. The same amount carried within the left for two days relieved all symptoms. Extraction of impacted teeth advised and proper balancing of occlusion.

Group V. Cases originating in actual destructive lesions within the temporomandibular joint:

CASE 19. (167.) Mrs. C. H., age 37, February 10, 1937. (Orbital pain, left; trismus; postdiphtheritic paralyses of all voluntary muscles, left side of the face, jaw, neck, and tongue.)

Above muscles paralyzed since diphtheria at the age of 12, and for the past year has had pain in the left cheek radiating to the left eye. Patient guarded against wide opening of the mouth, because this movement of the jaw produced the pain strongly.

Positive findings: The head was held tilted to the left about ten degrees off of horizontal and the tongue protruded to the left when extended. Mouth opening limited to one inch but can be forced beyond this. All molars missing except left lower. The left mandibular joint was quite tender to palpation. X-rays showed joint structures normal except that the left was fixed on opening flush against the tubercle as if the disc were worn; general fibrosis of the left joint capsule.

Course: Treatment dentures supplied to molar spaces, completely relaxed the trismus and relieved pain; after six months these were replaced by permanent dentures with remarkable improvement in paralytic jaw movements.

CASE 20. (174.) Mrs. C. H., age 54, April 26, 1937. (Trismus; left aural pain; malocclusion.)

Severe pain deeply about the left ear, past six months, worse during jaw movement and toward the end of the day—crackling noises in the left ear.

Positive findings: Very poor occlusion with uneven contacts made worse by poor dental construction. Trismus limited mouth opening to one-half inch and mandibular joints quite tender. Patient tilted the head to the left to accommodate a strong right rotary nystagmus.

Course: Pain and trismus definitely relieved by resting 4 mm. cork within the left jaw for half an hour. Full mouth reconstruction was done by numerous extractions, occlusion balanced with partial dentures. Relief of symptoms in six months.

CASE 21. (253.) Mrs. A. B., age 73, October 1, 1938. (Trismus; tinnitus left; aural pain left; erosion of left mandibular joint.)

Failing vision for many years with long history of sinus surgery, radium treatment, pelvis, etc. Past few years had ringing noises and pain within the left ear, increased by a yawn, and painful spasm of the muscles of the jaw. The trismus lasted several hours when induced.

Positive findings: Mouth overclosed widely on 30-year-old plates, with a snap of the condyles to the left. Left mandibular joint extremely tender. X-rays showed advanced erosion of the left condyle with narrowing of joint spaces.

Course: Patient greatly relieved by resting 4 mm. cork within both sides for a few minutes. Advised to replace dentures with ones giving adequate support.

CASE 22. (260.) Mrs. H. S., age 55, November 11, 1938. (Right aural and orbital pain; trismus; tinnitus right.)

During past month only, after strenuous times with tragedy in the family, patient developed pain deep within the right eye and ear. She had had crackling noises in the ears for years and tinnitus on the right side. Dental plates had been worn for nine years and her dentist recently opened the incisor dimension of her bite by appliques, greatly increasing her pain.

Positive findings: Left mandibular joint extremely tender to palpation with crepitation on opening and closing. Trismus varied

from complete spasm when jaw is manipulated to opening of about one-half inch. X-rays showed both condyles resting so snugly within the glenoid fossi that very little joint space remained. The right condyle was partially fixed on opening, against the tubercle, as if from trismus.

Course: Greatly relieved by carrying 2 mm. cork within the left molar district three days. Advised new dental plates, closing the incisor district slightly and extending vertical dimension on the left.

CASE 23. (264.) Mrs. E. H., age 71, November 26, 1938. (Trismus; general neuralgia, right side; glossodynia, right.)

Burning of dorsum of tongue past four years with pain to right side of nose, right eye and vertex, so intolerable past year that she had refused to carry her dental plates. Mouth dry most of the time. Spent most of waking hours moaning with pain.

Positive findings: Mouth edentulous, patient resisting the effort to open her jaws because of pain. Manually moving the lower jaw forward and held for five minutes greatly relieved the pain above described. Extensive geographic lesions on dorsum of tongue with large area exfoliated and several deep fissures. X-rays showed marked erosion of the face of the left condyle which rested against the tympanic plate in the closed position and moved forward a fraction to the middle of the glenoid fossa on opening, showing fixation; the right condyle fitted snugly in the glenoid fossa on closure and impinged the bone of the tubercle in the open position.

Course: After demonstrating the increased comfort to the patient from change of the jaw position, she was referred for test treatment with new dentures. At first these were measured to increase slightly the molar vertical dimension, closing the incisor district slightly. During the first few days of the test the patient was given small doses of codeine and the lower jaw supported with elastic headgear. She was completely relieved of pain after two weeks. Several recurrences of pain were relieved when the patient fixed the lower jaw three to four hours daily with the elastic jaw support.

SUMMARY

1. The frequency with which trismus was relaxed by a slight change in molar support indicates that abnormal condyle pressure within the glenoid fossa is the means of accentuating and continuing the trismus reflex; and sensory irritation of the auriculotemporal

nerve may be reflected to the motor portion, the masticator nerve, tensing the muscles of mastication.

2. Trismus may be gradually produced by the subpainful stimulus supplied by impacted teeth, root abscesses or caries, with or without inflammatory reaction.

3. The trismus effect may be initiated in a loosened mandibular joint with no evidence of bone changes, or in a joint showing erosion of bone, or by various sudden unusual movements of the lower jaw.

4. Trismus which at first is relaxed on movement of the condyles away from pressure areas by temporary support within the molar district, may be permanently relieved when the sources of the reflex, such as infection, impactions, or caries have been removed.

5. The results of x-ray study of mandibular joints in trismus, show (a) asymmetry of joint spacing, (b) limitation of the forward excursion of one or both condyles along the articular tubercle, (c) occasional cases with unilateral trismus on the side opposite the lesion (result of voluntary guarding of the painful side).

6. A most important feature in treatment is the fixation of the lower jaw by an elastic chin strap, and its leverage on the temporary or permanent molar support.

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XLIII

SERIOUS COMPLICATIONS OF FRONTAL SINUS DISEASE*

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OMAHA

Titles of papers on medical subjects often, like newspaper headlines, fail to reveal what the writer wishes to express.

The purpose of this paper is to report three cases of paranasal sinus disease with orbital extension.

The orbit and retrobulbar area are not infrequently involved in inflammatory diseases of the paranasal sinuses.

Extension takes place either by thrombophlebitis of the small veins leading into the orbit, or by direct extension through the walls of the orbit. In the latter event a periostitis of the orbital periosteum with or without subperiosteal abscess formation may occur, and if a subperiosteal abscess ruptures into the orbit an orbital abscess results.

Necrosis of the bony floor of the frontal sinus, the roof of the maxillary sinus and the external wall of the ethmoid or sphenoidal sinuses may extend through the somewhat resistant periorbital tissue and produce an orbital cellulitis or an orbital-phlegmon; the latter a most serious complication.

Transmission of inflammatory processes from the paranasal sinuses to the orbit by way of venous anastomoses occurs perhaps the most frequent.

The infection causes a thrombosis of the perforating veins, which leads to a local metastatic inflammation on the orbital side of the bone and from this further extension of the inflammation takes place.

The venous plexuses of the various sinuses frequently connect by perforating veins with either the inferior or superior ophthalmic vein or both.

The numerous venous anastomoses between the orbit and the lining membrane of the paranasal sinuses explain that, under condi-

*Presented before the Middle Section of the American Laryngological, Rhinological and Otological Society, Sioux City, Iowa, January 19-20, 1939.

tions even without direct extension, orbital complications may occur from inflammatory paranasal sinus disease by way of the veins alone, just as intracranial complications frequently do.

It is surprising, with the intimate anatomic relations between the paranasal sinuses and the orbit, that the number of orbital complications of sinus disease is not larger.

Orbital complications are usually associated with acute inflammations or an acute exacerbation of a chronic paranasal sinus inflammation and are especially associated with influenza, measles and scarlet fever.

The number of sinus cases with orbital complications caused by swimming and diving is striking.

The first sign of orbital extension is usually a swelling of one or both eyelids, this may or may not be associated with chemosis of the palpebral conjunctiva.

As the intra-orbital pressure increases, the movements of the eye become limited and the globe becomes displaced. The direction of displacement depends somewhat on the sinus involved. That is, in orbital extension from the frontal sinus, the eye should be displaced downward and outward; whereas, in extension from the ethmoid the displacement is more likely to be outward.

Where the maximum swelling and pressure is posterior to the equator of the eyeball, as in extension from the sphenoid, an exophthalmus results.

Severe pain and marked tenderness in and about the eye and sinus involved is always present. The temperature is elevated, the degree depending upon the severity of the infection and whether or not a thrombophlebitis is present.

X-ray examination of the sinuses is a valuable diagnostic aid, inasmuch as intranasal examination often shows very little.

The following cases present some interesting problems on this subject:

REPORT OF CASES

CASE 1.—L. D., female, age 35. Patient had a severe head cold for ten days, then developed a severe frontal headache on the left side. The pain soon became unbearable and morphine was administered frequently. A swelling appeared in the left upper eyelid, which quickly spread to the lower lid. Temperature ran around 102 de-

grees. There were no chills. She was sent to Omaha and entered St. Joseph's Hospital, February 23, 1938.

Examination: Due to sedatives the patient showed sluggish, atonic reactions and her speech was slurred.

The left upper and lower eyelid were markedly swollen and reddened and the conjunctiva was chemotic. The eyeball was fixed. A contracted pupil reacted slightly. Fundus showed no great venous engorgement. Slight edema of the disc was present. Neurological examination showed no other cranial nerve involvement, no neck rigidity, no Babinski, no paralysis.

X-ray shows moderately cloudy left frontal, maxillary and ethmoid sinuses.

There was a small amount of purulent secretion in the left middle meatus.

The temperature was 103 degrees; white blood count, 24,000 cells, 80 per cent of which were polymorphonuclears.

This patient presented the picture of a beginning cavernous sinus thrombosis. Marked edema of both upper and lower eyelids, chemosis of the conjunctiva and fixation of the eyeball. Miosis of the pupil, engorgement of the retinal veins and septic temperature with chills were, however, not present.

Dr. J. J. Keegan, a neurological surgeon, was of the opinion that the frontal and ethmoid sinuses should be drained, even though there was a possibility of thrombosis of the cavernous sinus.

An external frontal and ethmoid operation was done that night and a serious prognosis given. The frontal sinus and ethmoid cells were filled with pus and hyperplastic membrane. There was no bone necrosis of the frontal sinus or the orbital plate of the ethmoid.

Complete recovery took place after four weeks.

The diagnosis of beginning cavernous sinus phlebitis was made on the presence of paralysis of the fourth and sixth nerves, which produced fixation of the eyeball, and the swelling of the eyelids, with chemosis of the conjunctiva. Thrombosis with bloodstream pollution probably was not present. The organism recovered was staphylococcus hemolyticus.

CASE 2.—L. R., male, age 14. Case of Dr. F. M. Watke. A young boy who frequently went swimming, complained of a severe right frontal headache for about ten days, then a swelling of the

upper right eyelid appeared. The eye became proptosed. Temperature ran about 100 degrees, and there were no chills.

The writer saw the patient in consultation July 9, 1938, five days after swelling appeared. At this time there was a marked swelling of the upper right eyelid with some chemosis of conjunctiva. The eyeball was partially fixed and proptosed. There was marked tenderness over the right frontal sinus. There was no papilledema or engorgement of the retinal vessels.

Very little secretion was present in the nose in spite of continuous hot packs and intranasal tampons.

X-ray: Clouding of right frontal, ethmoid and maxillary sinuses. White blood count 12,000 cells, 75 per cent polymorphonuclears.

A diagnosis of right pansinusitis with possible orbital subperiosteal abscess was made and external operation on frontal and ethmoid sinuses advised.

The evening before the day set for operation, there was a profuse discharge of blood and pus from the right nares, and marked improvement in all signs and symptoms followed.

Complete recovery, without operation, rapidly took place.

A diagnosis of a subperiosteal abscess of the orbit was made because of the orbital findings and lack of severe systemic symptoms.

It is supposed that the abscess ruptured through the ethmoid into the nose. Recovery took place so rapidly that this conclusion seems logical.

CASE 3.—R. M., male, age 12. Case of W. P. Jensen. This boy was also a frequent swimmer, spending two or three hours a day in the pool.

One Sunday evening he was seized with a terrific frontal headache, accompanied by nausea and vomiting. This continued for thirty-six hours, then he was brought to the hospital.

Examination August 23, 1938, the day he arrived in hospital: Patient in comatose condition with flaccid paralysis of right extremities and complete paralysis of right facial nerve. There was no neck rigidity or Koenig's sign. There was some edema and ptosis of the upper left eyelid. Chemosis of the conjunctiva was not present. No fixation of the eyeball and the fundus was normal. A small amount of purulent secretion appeared in the pharynx but none was seen in the nose.

X-ray: Left maxillary and ethmoids slightly cloudy. Frontals clear. White blood count, 27,000, 88 per cent polymorphonuclears.

Spinal puncture was done and the fluid was cloudy. Pressure 7 mm. No increase in pressure when left jugular was compressed. Pressure increased to 12 mm. when right jugular was compressed. The cell count on the first spinal tap was fifty. Temperature 104 degrees.

By five o'clock in the afternoon the paralysis, which was present that morning, had disappeared, though the boy was still irrational. Spinal puncture was done daily and the cell count reached 210, and became normal after five days.

The swelling of the upper eyelid spread to the forehead toward the center and disappeared from the eye. This swelling increased in size, became quite soft and was opened September 9th. A small amount of blood and pus was evacuated and the frontal bone under the abscess was roughened.

The parents refused any operative procedure on the sinuses or skull, inasmuch as the boy had shown marked improvement in general.

He was discharged from the hospital September 11th, apparently recovered.

On November 11th, for the purpose of this report, an x-ray was taken, and the film shows a large area of necrosis of the frontal bone with sequestration.

This case presents rather unusual conditions; hemoplegia lasting about eight hours with rapid recovery and meningitis.

There is little question but that infection extended in this case by way of thrombophlebitis of the veins leading into the orbit and through the orbit to become an intracranial infection.

The sudden appearance of intracranial symptoms and osteomyelitis of the frontal bone beginning some distance from the frontal sinus bespeaks a thrombotic process.

Although this patient is apparently in good health at this time, there is a possibility that the process in the frontal bone may light up at any time with serious results.

A similar case seen several years ago died of a brain abscess about a year after recovery from the initial sinusitis and osteomyelitis. Good health was enjoyed in the interim.

COMMENT

The three patients had one thing in common, that is, orbital extension from acute paranasal sinus disease.

In the first and third case, the infection probably extended by way of the blood vessels, and in the second case by direct extension through the orbital wall.

Treatment in the beginning was the usual local and symptomatic treatment of sinus disease. Sulfanilamide was given in all cases and may have been of benefit to the boy with meningitis.

In the treatment of this type of case, where involvement of the orbit has taken place, surgery should not be delayed too long.

In spite of the possibility of extension of an osteomyelitis after operation, free evacuation of pus from the diseased sinus should be accomplished.

The boy whose abscess ruptured spontaneously into the nose was fortunate, because the abscess could have ruptured into the orbit and set up an orbital abscess.

In conclusion, those patients who develop orbital extension from sinus disease by way of the blood vessels, present more difficulties than those who have a contact extension, because there is less time for local resistance to be set up.

536 City National Bank Bldg.

XLIV

PRIMARY SQUAMOUS CELL CARCINOMA OF THE UVULA: REPORT OF TWO CASES*

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AND

Merrill W. Michels, M.D.

Detroit

Primary squamous cell carcinoma of the uvula occurs so rarely that such cases should be reported. Intra-oral tumors constitute about three per cent of all neoplasms. The most complete series reported to date is that of Stout,[1] who found only twenty-eight cases of tumor of the uvula over a fifty-year period. Of these, nine were carcinoma, but microscopic examination was reported in three instances only. Since then occasional isolated cases have appeared in the literature (Kahn[2] and Howarth[3]). In a series of 2,471 cases of intra-oral cancer reviewed by Quick,[4] no mention is made of primary tumor of the uvula, although neoplastic growths of the hard and soft palate occurred in 106 cases.

The symptoms noted with tumors of the uvula are usually referable to the local process. Pain may be present in a circumscribed area or may be diffusely spread throughout the entire oropharynx, often radiating to the ears. Difficulty in swallowing, dryness, hoarseness and tickling of the throat are occasionally manifested.

Surgical exenteration of the early malignant process prior to metastasis to the upper cervical and sternocleidomastoid chains of lymph nodes apparently offers a better prognosis than irradiation alone. According to Cutler,[5] carcinomas arising in the pharyngeal pillars and soft palate are differentiated infiltrating carcinomas which are locally destructive and very difficult to sterilize by irradiation. Carcinomas of the anterior pillar and palate, in contrast to malignant lesions of the tonsil, have a poor prognosis because of the difficulty in sterilizing the primary lesion. Irradiation therapy is thus largely palliative, for external irradiation is rarely able to sterilize carcinoma of the anterior pillar, soft palate or angle between the pillar and the base of the tongue.

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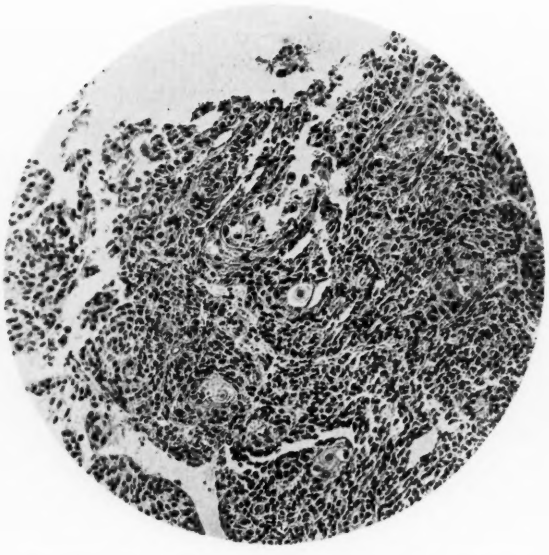

Fig. 1. Squamous cell carcinoma of the uvula with beginning pearl formation. (Case 1.)

REPORT OF CASES

CASE 1.—J. N., a 39-year-old white male, was admitted to the Out-Patient Department on September 1, 1938, complaining of intense soreness and burning throughout the entire mouth. The symptoms had been present for three months and had become so intensified that he was unable to swallow food or fluids. He was a moderate smoker and had a negative past history except for a twenty-pound weight loss. Examination revealed a moderately enlarged uvula which had a verrucous appearance. There was apparently no extension beyond the base of the uvula or upon the velar arch. The teeth were in a poor state of repair; a well advanced pyorrhetic process was present. No cervical glands were palpated. Systemic examination was otherwise negative except for malnutrition.

The base of the uvula was infiltrated with one per cent novocain, and by means of a wire snare the uvula with a generous portion of soft palate was excised for microscopic examination. The section showed reticular masses of epithelial cells with beginning pearl formation characteristic of a squamous cell carcinoma. (Fig. 1.)

Fig. 2. Squamous cell epithelioma of the uvula with considerable anaplasia but no diffuse infiltration of the surrounding tissue. (Case 2.)

The patient was admitted to the hospital and resection of the soft palate was performed by electrocautery. Microscopic examination of this specimen revealed chronic inflammation and edema with no extension of the neoplastic process. The patient had a period of regurgitation of fluids through the nose following operation but regained control within a month.

Supervoltage x-ray therapy was administered with 500 K.V. equivalent (7 mm. cu.). The dosage amounted to 170 per cent S.U.D. accumulated over a period of twenty-two days (October 17, 1938 to November 5, 1938). One portal of entry was used, alternating each side of the face and neck with daily fractions of 200 r. (in air) on the skin. To date the patient has been in good health with no evidence of metastasis.

CASE 2.—E. M., a 53-year-old white male, was admitted to Harper Hospital on January 19, 1939. He had been in excellent health and had no complaints referable to his mouth lesion. His dentist had discovered a small growth of the uvula; the total duration

of symptoms probably did not exceed one month. He had no dysphagia or local pain.

The uvula was enlarged to the size of a raspberry and was very similar in appearance with numerous small rugose nodules over its surface. There was no apparent extension to the adjacent palate. The mouth hygiene was poor; two unsatisfactory bridges were noted. No glands were palpated in the submaxillary region or posterior cervical triangles. Physical examination was otherwise negative.

Surgical exenteration of the uvula was performed under local anesthesia by means of a Tyding's tonsil snare. A generous portion of the soft palate was included. Microscopic examination of this section showed a squamous cell epithelioma with considerable anaplasia but no diffuse infiltration of the surrounding tissues. (Fig. 2.)

The patient was discharged on the day following his operation and has remained in good health.

CONCLUSIONS

In conclusion, two cases of primary squamous cell carcinoma of the uvula are presented. Such malignancies are of rare occurrence. Early excision offers a better prognosis than irradiation therapy alone in differentiated infiltrating types of neoplasms.

641 David Whitney Bldg.

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XLV

SPONTANEOUS HEMORRHAGE FROM THE EAR

THOMAS F. MCCORMICK, M.D.

MILWAUKEE

Spontaneous hemorrhage from the ear is apparently such an infrequent complication that the standard ear texts make little or no reference to it. Politzer, Koerner and Jansen mention it in connection with tuberculous erosion of the carotid, and Denker speaks of it only following injuries to the jugular bulb in paracenteses; he mentions six recorded cases. In an old text of Politzer's (1882), there are cited thirteen cases of fatal bleeding from the ear due to involvement of the internal carotid, all in chronic suppurations and seven of them tuberculous. Salinger and Pearlman's painstaking and complete resume of the literature lists 20 cases of spontaneous aural hemorrhage resulting from parapharyngeal infection. Of these, 16 were fatal and autopsies in all except one showed erosion of the internal carotid. This one case, that of Riecke, exhibited no involvement of the great vessels of the neck, the probable source of bleeding being from the internal jugular near the bulb. A pertinent observation regarding the ages of these 20 patients is that spontaneous bleeding from the ear is invariably confined to children. The oldest in the group was 14, one was 13, one 11, one 9, and twelve were 4 years or under.

REPORT OF A CASE

CASE 1.—Richard H., age 2, previously healthy, was seen at home ten days after the onset of illness. This began with a sore throat, fever, and within a few days palpable glands on both sides of the neck. For the preceding 24 hours he had been very irritable and pulled at his ears.

Examination showed a mass of glands at the angles of the jaw on both sides, and the throat moderately injected but with no evidence of abscess. The right drum was reddish gray and bulging in its posterior half; this was incised. The left showed a pale diffuse blush over its entire surface with the light reflex still present.

On the following day the right ear presented a moderate amount of drainage, the left a very profuse serosanguineous secretion; this drum membrane had opened spontaneously early in the morning. At

5:30 that afternoon, while being held in his mother's arms, there was a sudden violent hemorrhage from the left ear, which was controlled (or stopped spontaneously) after packing the canal. The amount of blood lost was around 500 cc. The patient was taken at once to St. Mary's Hospital and given a transfusion of 300 cc. of blood.

At intervals of about 12 hours for the next two days the bleeding recurred but not to the alarming degree as on the first occasion. The canal was kept tightly packed and the dressings changed every 24 hours or oftener. On the morning of the third day the entire bandage suddenly became bloody in only a few minutes, and as on the other occasions the bleeding stopped spontaneously.

Meantime another transfusion of 250 cc. had been given. His condition remained good, he took fluids well, his temperature ranged between 101 and 103.5 degrees, and until this last bleeding his pulse was under 140. Seen shortly after this incident his pulse rate rose to 160 and he was very restless and irritable. An x-ray film of the ear showed only some indistinctness of cell outline; because of the voluminous bandage an accurate interpretation was impossible. The throat was entirely symptomless and the adenitis unchanged.

Our feeling, whether justifiable or not, was that the bleeding was from the neighborhood of the jugular bulb; so that day, the third following admission, the mastoid was opened and cleaned out, the lateral sinus exposed over an area of about 2½ cm. and packed off at the upper and lower ends by introducing half-inch gauze packing between the sinus wall and the bone, using enough gauze to insure complete collapse of the walls. There was some breaking down of the cells in the region of the tip, the remainder showing no bony destruction. The sinus itself was entirely normal in appearance. Another 300 cc. of blood was given at this time.

There was no further bleeding, and except for a few minor incidents the postoperative course was satisfactory. The packs compressing the sinus were taken out on the fifth and sixth days, the walls remaining collapsed after their removal.

In view of a study of the literature, there is certainly just cause for criticism in the treatment of any case of alarming bleeding from the ear if the common carotid is not immediately ligated. While the amount of blood lost here was considerable each time, and on the occasion of the final bleeding was such that the child was almost exsanguinated, the ear symptoms were predominant, the adenitis showing no progression and the throat no evidence of the presence

of pus. There was no bleeding from the mouth or nose at any time, the inflammatory process in the middle ear apparently having closed off the eustachian opening, nor was there evidence of any break in the lining of the external auditory canal. Had the drum on this side, the left, been incised the hemorrhage would with certainty have been ascribed to a wound of the jugular bulb.

This case is reported because it is an unusual one, in that any profuse spontaneous bleeding from the ear is unusual. The apparent source of bleeding, from the bulb or the jugular vein near the bulb, is also unusual, and, finally, the procedure employed to control the hemorrhage is at least out of the ordinary.

1141 WELLS BUILDING.

XLVI

REPORT OF A CASE OF SPONTANEOUS CEREBROSPINAL RHINORRHEA WITH OPERATIVE CURE

ANDREW WESSELS, M.D.

SAN ANTONIO, TEXAS

CASE 1.—Mrs. M. G. B., aged 49, consulted the writer on March 2, 1938, for a persistent discharge of fluid from the left nostril. There was a history of headaches beginning about eight years ago, mild at first and lasting a short period of time, later becoming severe and persisting from twelve to twenty-four hours. Sleep at first brought relief, later on did not. There was impairment of vision, slight to begin with, and later enough to interefere with her work in her office. She was refracted several times during the first few years, with no apparent relief from the wearing of glasses.

The headaches were accompanied by a burning sensation in the right eye, pain in the right temple, right mastoid, right occipital region and the right deltoid muscle, occasionally in the right elbow.

There was no disturbance of vision except during the time she had these headaches. During the first half of 1937 the headache was almost constant and the vision was markedly affected in the right eye only. She was given tuberculin and vitamin B for several months, during which time the headaches became less severe, with an alleviation of the other symptoms and improvement in vision.

There is no record of examination of the fundus of either eye during this period.

About the middle of July, 1937, while at work in her office, there was a sudden flow of fluid from the left nostril. She felt "feverish" and quite weak and was unable to work. She remained in bed about a week before returning to her office. The nasal discharge was constant for about eight months before she consulted the writer. The amount of fluid which dropped from the left nostril with the head held forward varied from 10 cc. to 15 cc. in thirty minutes. It was a clear, yellowish fluid, showed negative complement fixation test on five dilutions and the control Kahn was also negative. Sugar, 54.05; N. P. N., 14.23; chlorides, 627.0 mgm.;

total protein, 14.5 mgm. per cent. There was no question about the origin of the fluid.

Several radiographic studies showed only a suggestive haziness of the frontal sinuses, from a plus 1 to a plus 2 involvement of the anterior and posterior ethmoid sinuses. The left maxillary showed almost a plus 3, while the right maxillary showed a plus 2 hyperplasia of the lining membrane. The sella turcica was large, there was a thinning of the posterior clinoid processes, the intersphenoidal septum could not be visualized, and the lateral walls of the sphenoid sinuses were not well defined. The radiologist suggested that there might be an intracellar tumor in the middle fossa.

The eye findings showed only a few fine vitreous opacities in the right. The discs were of good color but the disc margins were uneven and indistinct and the disc itself was surrounded by a narrow whitish band. Visual fields showed some loss of vision for white in the temporal field in the left eye. The oculist's comment was, "That some time in the past there had either been a choked disc or papillitis, although at present the discs are not elevated."

No intranasal instrumentation or medication was attempted for fear of initiating an ascending infection. The patient was referred to Dr. Ernest Sachs of St. Louis, who made an encephalogram followed by a cerebral craniotomy on April 8, 1938, and noted the following: "Encephalogram done under avertin. The plates threw no light on the lesion. In view of the leak through the left nostril I did a left frontal exploration of the chiasmal region. The left optic nerve was readily exposed and appeared normal. The right optic nerve could be clearly seen. The left and right internal carotids could be seen and there was a sclerotic plaque in the right internal carotid artery. There is no evidence of a pituitary tumor, no evidence of the cerebrospinal fistula. The left olfactory nerve was cut and the dura was stripped up from the cribriform plate on the left side and a depression in the anterior fossa was clearly seen, the cause of which, however, was not apparent. At one spot the bone seemed to be completely destroyed, and it may be that this is where the cerebrospinal leak was taking place. A piece of temporal muscle was therefore excised and packed over this area. The dura was then opened over the anterior portion of the frontal lobe and readings were taken from the cortex at three different points. The dura was then closed with interrupted silk and the cavity filled with saline. Then the bone flap was replaced and the periosteum closed throughout. Silk for the galea and skin and a drain was

inserted near the posterior end of the flap." Dr. Sachs was somewhat puzzled by the findings and could not understand the presence of the saucer-shaped cavity in the anterior fossa.

The patient has been under constant observation since her return. About October 1, 1938, she had a slight headache. She was refracted and her correction was changed and the headaches have entirely stopped.

She was operated upon on April 8, 1938, which is almost a year ago. There has not been any suggestion of a discharge from the nose or from the postnasal space since that date.

1705 Nix Professional Building.

Society Proceedings

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, December 5, 1938

THE PRESIDENT, DR. THOMAS W. LEWIS, IN THE CHAIR

Rhinoscleroma: Histologic Changes After Teleradiation; Review of Scleroma in the United States

J. A. WEISS

(Author's Abstract)

Scleroma was originally endemic in central and southeastern Europe. Since the Great War it has spread throughout the world. In the United States sixty-one cases have been reported in the past forty-five years. Eight of these were native-born Americans. It is possible that the disease is becoming indigenous.

Scleroma is a chronic specific granuloma characterized by an extremely indolent clinical course, a distinctive microscopic structure and a probable infectious etiology. The lesions usually start in the nose and progressively involve the nasopharynx, oropharynx, larynx and trachea. Less often extension occurs to the upper lip, tongue, orbit, lacrimal apparatus and bronchi. Primary laryngotracheal cases occur.

Three stages of the disease are described: First, the diffuse, catarrhal stage; second, the circumscribed nodular stage, and third, the cicatricial stage. The symptoms depend on the functional impairment of the area involved. There may be nasal discharge or occlusion, dysphagia, hoarseness, cough or dyspnea.

The diagnosis is based on the occurrence—usually in the foreign-born—the great chronicity of the course, the hardness of the lesions, the absence of pain, ulceration or systemic involvement and the eventual cicatrizing deformities. Frisch's bacillus may be found in culture. A specific serologic reaction and an intracutaneous test are usually positive.

Scleroma must be distinguished from papilloma durum, syphilis, lupus vulgaris, leprosy, sarcoid (Boeck), carcinoma and sarcoma.

Irradiation and vaccinotherapy combined with essential surgery or electrocoagulation offer the best methods of treatment.

Two cases are reported. In the first a small nodule was excised and 20,000 mgm. hours of distance radiation was given with a 2 gm. radium pack. There was no recurrence in one year. In the second case obstructive lesions in the nares regressed after exposure to 32,000 mgm. hours of radiation. The stenosis of the nares was corrected surgically. There has been no recurrence in two and a half years.

The microscopic structure of scleroma consists of a thin connective tissue reticulum with a hyperplastic epithelium and a stroma of cells of chronic inflammation including the typical vacuolated Mikulicz "foam" cells and the Russell hyaline bodies. The changes after radiation consist in (a) extensive fibrosis; (b) increased hyalinization; (c) disintegration and disappearance of the Mikulicz cells.

Teleradiation in higher dosages will eradicate circumscribed scleromatous lesions and probably prevent recurrences.

DISCUSSION

DR. SAMUEL J. PEARLMAN: The essayist has given us a very clear and concise picture of rhinoscleroma, which, in this country, of course, is not very common. I do not believe I have ever seen a case here, although I saw some in Vienna. There are many men who spend a lifetime in the practice of laryngology and never see a case. Out of the 58 cases reported in the American literature there are some eight that were indigenous. They occurred in individuals who spent all their lives in the United States, and this may be a small arrow pointing in the direction of more and more cases occurring in natives of this country. From a practical standpoint, while not a very common disease, it must be borne in mind and must be differentiated from those cases the essayist told us about, syphilis and tuberculosis. I was pleased to see here for the first time described in the literature the histology of rhinoscleroma following radiation therapy.

DR. JOSEPH C. BECK: Some twenty-five years ago the late Dr. Stanton Friedberg and myself had at the Cook County Hospital a

case of rhinoscleroma in a Rumanian girl aged 19. The reason I rise to discuss this paper is because I failed to hear mention made of the extension into the esophagus. Our case required a gastrostomy on account of the inability to obtain a passage large enough to pass a feeding tube through the esophagus. Of course, you realize that at that time we had no such treatment as irradiation. There were, however, very noticeable effects in that case when we administered large doses of potassium iodide, of which I also failed to hear any mention made. It was recommended as the orthodox method of treatment before irradiation and vaccine therapy. Our patient was discharged from the hospital with a permanent gastrostomy; nothing further was done.

DR. S. A. SCIARRETTA: I have enjoyed Dr. Weiss' presentation. While listening attentively, two important points occurred to me. The first was the symptomology, which in the two cases presented were typical of this malady. In the Bollettino Delle Malattie Dell'Orecchio, Della Gola, Del Naso, October issue of 1937, Simonetta reported six cases of scleroma. All presented symptoms of obstruction caused by the scleromatous growth located in the uppermost portion of the trachea or in the larynx and its surroundings and in the nasopharynx. While these tumefactions created scar tissue, adhesions and destruction of anatomic landmarks, the nasal symptoms in practically all cases were of the mild type; that is, copious and foul watery secretion with crusting, as described by Dr. Weiss to be the early warning signs. In other words, the early symptoms in these cases represent the late symptoms of scleroma as described in books. The therapy used by this writer consisted of teleradiation, injection of gold solutions, and electric coagulation when indicated. His results were good, but I think there were some recurrences.

The second point was the up-to-date, concise review of the histologic structure of scleroma presented by Simonetta. Streit studied these characteristic cells in 1914, and his complete work appears in the Denker and Kahler textbook, 1928 edition. In 1935, Streit described two distinct types of Mikulicz cells and stated that while they may originate from various types of tissue cells, he believes they really derive from the epithelial cells. Dr. Simonetta agrees with Streit in differentiating these cells, but as to their origin, he claims that the majority of typical Mikulicz cells develop from the connective tissue cells. He is convinced that few of these cells develop from the epithelial cells and have a polyhedral or oval form, a clear center and some residual protoplasm on one or both poles. The typical cells described by Mikulicz derive from leucocytes

which, during the developing process of scleroma, invade the epithelial tissue; later they degenerate and then appear as large vacuolated cells with a distinct capsule at the margin of which a pyknotic nucleus, or fragments thereof, is visible. I think Dr. Weiss has covered the subject thoroughly.

DR. HANS BRUNNER: Cases of rhinoscleroma were seen more frequently in Austria when the eastern parts of Europe (Poland and Bukowina) were under the authority of Austria. Since the World War, however, there has been a considerable decrease in the incidence of this disease in the clinics of Vienna. Acute cases are rarely seen at the present time. Instead, only far advanced cases with scar formation and stenosis are observed.

DR. WALTER H. THEOBALD: I rise not to discuss Dr. Weiss' paper but to compliment him on his very excellent and concise presentation. He did very thorough and careful work in preparing the histologic part of his paper. I mention this because I want to call to your attention that this is a candidate's thesis. It was unanimously recommended by the Thesis Committee that this paper be read, and it is one of the few theses to be read before the Society.

DR. J. A. WEISS (closing): I wish to thank the gentlemen for contributing their discussions to the paper and Dr. Theobald for his very kind words. His graciousness is exceeded only by his skill as an otolaryngologist.

The increasing incidence of scleroma makes its recognition of practical importance. In reviewing the cases in the literature one finds that laryngeal scleroma is frequently mistaken for syphilis.

The Air Cells in the Petrous Temporal Bone in a Child of 13 Years

(Lantern Demonstration)

J. GORDON WILSON

(This presentation was presented in full on page 35 of the March, 1939, issue.)

DISCUSSION

DR. BARRY J. ANSON: I wish to comment on Dr. Wilson's paper by presenting, for comparison, the anatomy of the apical cells in a younger child, aged 3½ years. I shall demonstrate with lantern slides the topography and shape of the air cells at the petrous tip,

selecting four illustrative levels. In this specimen the tympanic orifice is similar in form and location, but distributed more than in the preceding case. For this specimen we have wax-plate reconstructions, prepared from the serial sections.

Section 1. Through the cranial opening of the carotid canal, the upper portion of the cochlea and of the superior semicircular canal. They are near, but not continuous with, the epitympanic and mastoid cells which surround the superior semicircular canal; they do not reach the internal acoustic meatus.

Section 2. Through the vestibular (oval) window, ossicles and the auditory tube. Cells at this horizontal level are at their maximal extent; as yet they are without communication with the tympanic cavity (middle ear); the cells pass, in an interconnected group, beneath the carotid canal to the cranial side of the petrous tip where they are separated from dura mater by a thin plate of bone (inner table).

Section 3. Nearing the floor of the auditory tube; through the tympanic membrane and the attached malleus, the fossa for the jugular vein. The apical air-cells open, by broadened communication (tympanic orifice or ostium) into the space of the middle ear.

Section 4. At the floor of the tympanic cavity, near the inferior attachment of the tympanic (drum) membrane. Here, close to their inferior limit, the air cells still open into the lowermost part of the tympanic cavity middle ear by several additional communications (comparable to nasal orifices or ethmoid cells). They lie in walls of the canal for carotid artery and the fossa for the jugular vein.

Reconstructions: As verified graphically in reconstructions of the air cells and associated structures, the cells extend upward, arch over the carotid canal to reach its cranial side; anteriorly they extend toward the cochlea, while posteriorly they pass along the carotid canal, then beneath the latter; they communicate with the tympanic cavity by a definite orifice or ostium, situated at the horizontal level of the vestibular window; they communicate additionally by openings situated on the floor of the cavity, located near the opening of the auditory tube. The two large groups of cells (passing over and beneath the carotid canal) approach each other, yet do not actually meet on the cranial side of the canal; they surround the carotid canal almost completely. In general, the cells resemble those of the mastoid portion of the temporal bone. The greater complexity in form of

the apical cell group is seemingly due to the presence of the carotid canal; encountering this obstacle in the course of their invasion of bone they pass along, under and above it, pneumatizing its bony wall.*

DR. S. A. SCIARRETTA: I wish to call attention to another work on this subject written by Federici of the University of Genova and published in the Archivio Italiano di Otologia, July, 1936. He reported a very interesting case, operated on several times without much improvement. Finally, when the peritubal air cells were discovered to be infected and were opened, the patient made an uneventful recovery. In this literary review, the author points out the numerous classifications advanced by many observers. He concludes that the various groups of air cells found in the temporal bone should be separated into two distinct divisions.

The cells located anteriorly and inferiorly to the petrosal pyramid should be called the bullar air cells; in this group he includes the subcochlear, peritubal and pericarotid air cells. I think this is the group of air cells which Dr. Wilson has so masterfully described and illustrated to us this evening. Federici states that pathologic processes invading this group of cells invariably originate from the middle ear.

The second group of air cells comprises those which originate from the antrum and are located postero-superiorly and posterio-inferiorly to the pyramid; that is, the sublabyrinthine, supralabyrinthine and translabyrinthine air cells.

DR. ALFRED LEWY: I should like to ask Dr. Wilson or Dr. Anson if we are to understand from their presentation that there are only two origins for cells which appear in the petrous pyramid, one communicating with the tympanic cavity in the region of the eustachian tube, and the other group communicating with the epitympanic space. It has been generally held by clinicians that there are other groups of cells in the petrosa. I would like to ask whether those cells near the internal auditory meatus which can be reached from the posterior fossa are part of either one of these groups, or whether they constitute a separate group. If I understood Dr. Wilson correctly, he referred to those cells that surrounded the labyrinth as belonging to the epitympanic group.

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*Full description of anatomy in Archives of Otolaryngology, Vol. 27, May, 1938.

DR. ELMER W. HAGENS: I have enjoyed this presentation of Dr. Wilson and Dr. Anson very much. There is one point that I would like Dr. Wilson to bring out. He showed a very thickened tunica propria in the pneumatic space. I have a specimen of a child, age three years, with a pneumatic space in the petrous apex. In that space the tunica propria of the pneumatic space is very thick. I was puzzled about it, and explained it by saying that the tunica propria was thickened because the epithelium had not yet shrunk back and down to the bone, but in Dr. Wilson's specimen the child was 14 years old. If these pneumatic spaces develop early, by the fifth year even, and if it started at that age in his case, it must then take a long time for them to shrink. In adults we know that the epithelium is down practically to the bone. It is very interesting to find a child of 14 with pneumatic spaces developing and with the tunica propria so thick. Certainly at certain levels you see no lumen at all.

I have not had occasion to examine a child with pneumatic cells developed at that age, with such a thickened tunica propria. The cases that did, had infection and were explained on that basis. I would like to know whether Dr. Wilson has any idea of the length of time it takes for shrinkage of the tunica propria from childhood to the adult stage.

DR. J. GORDON WILSON (closing): Replying to Dr. Lewy, in the petrous temporal there are two cell groups with separate origins. Air cells lying along the wall of the tympanic cavity are not in groups similar to those in the mastoid or those I have described in the petrous pyramid.

As Dr. Hagens says, the tunica propria usually is thick at the age of three years. In the fetus, the middle ear is filled with embryonic mesenchymatous tissue which gradually shrinks, leaving clear areas in which the epithelium shows itself. This mesenchyme is replaced by fibrous tissue which extends to the antrum and helps in forming the air cells. After the first year, development goes on rapidly until the child is about five years old, after which it slows down. The appearance of thickness of the tunica propria in one of my sections is due to the plane of the section. Before the advancing connective tissue and epithelium helps to form the air cells, I believe there is softening of the adjacent bone. Osteoclasts are few and may be wanting. What happens to cell formation when the potential connective tissue and epithelium entering from the middle ear is altered by disease, either before or after birth, is disputed.

The Structure of the Ethmoid Labyrinth (Anatomic Study)

O. E. Van Alyea

(Author's Abstract)

The ground lamellae of the upper turbinates, the middle, superior and supreme, and the secondary turbinates, the uncinate process and the ethmoid bulla, pass laterally through the ethmoid mass to the orbit, separating the ethmoid cells into groups. Although these plates act as barriers to prevent intermingling of cells, they have very little control over cell expansion and commonly cells from one group invade the territory of another group. Invading cells, however, retain their original classification which is based upon the location of their drainage ostia in the nose.

Ethmoid cells tend to expand in all directions until stopped by hard, compact bone and often extend beyond the ethmoid field to adjacent structures, even at times passing through the nasal septum to project onto the structures of the other side. Cells commonly shell out the superior and supreme and attached portion of the middle turbinates and not infrequently grow into the pendulous portion of the middle turbinate; a bulky turbinate may be caused by two or three such cells. Of common occurrence also, are cells invading the frontal, maxillary and sphenoid bones. In an examination of one hundred specimens from cadavers, almost every form of cell encroachment both intra- and extramural was observed, and in this examination it was possible to determine the frequency with which the various types of encroachment occurred.

Ethmoid cells commonly drain into grooves. These form fairly parallel lines along the face of the upper lateral nasal wall. They are, in the middle meatus, the hiatus semilunaris and the bullar ostium (superior hiatus); in the superior meatus, the posterior groove; and in the supreme meatus, the postreme groove. One groove may accommodate the ostia of several cells or may be the wide drainage space of one cavity.

Dr. A. A. Zimmerman: As an anatomist I should like to commend Dr. Van Alyea for his scientific curiosity and his persistent effort in studying the anatomic relations and variations of the paranasal sinuses. It is always gratifying to see clinicians make extensive use of anatomic material. The greatest gain of such a study naturally is to the clinician himself and secondarily to his patients. Dr. Van

Alyea familiarized himself thoroughly, not only with the normal relations of the ethmoidal cells but with their multiple variations as well. On the basis of a sufficiently large number of specimens he was able to establish frequency tables of intra- and extramural encroachments. From the anatomic viewpoint these tables constitute a valuable contribution to our knowledge of the ethmoid labyrinth.

The nomenclature concerning the ethmoid labyrinth unfortunately is not uniform. Some authors would prefer to classify the ethmoid cells according to the bones which they invade (Poirier). In purely descriptive anatomy, particularly in osteology, such terms as fronto-ethmoidal, lacrimo-ethmoidal, maxillo-ethmoidal and spheno-ethmoidal cells are in use. Dr. Van Alyea justly avoided these terms. A rational grouping of the ethmoidal cells should be based rather on their origin. As the site of origin of any ethmoidal cell constitutes the normal ostium of the respective cell in the adult, such a terminology is of advantage to the clinician. This is the nomenclature usually employed in the description of recent specimens in which the mucosal lining of the cells or sinuses is intact.

It is generally agreed to designate as anterior ethmoidal cells the entire group of cells which is derived from the middle meatus or from its secondary recesses. The cells derived from the superior and supreme meati are grouped as posterior ethmoidal cells. Those originating from the supreme meatus sometimes are designated as postreme ethmoidal cells.

Seydel subdivided the anterior group into anterior ethmoid cells proper and into middle or bullar cells. Killian subdivided the anterior group into frontal and infundibular cells.

As a criticism of Dr. Van Alyea's paper, I should like to point out that he failed to consider the frontal ethmoidal cells. Although he mentioned this deliberate omission, it would appear to be hardly justified to exclude the frontal cells from an anatomic study of the ethmoid labyrinth. Specifically, the frontal ethmoid cells are derived from and therefore drain into the frontal recess. The latter is merely an antero-superior extension of the middle meatus proper. The group of infundibular cells, on the other hand, is derived from and drains into the infundibulum of the middle meatus. Dr. Van Alyea further subdivided this group. It should be kept in mind that all infundibular cells, disregarding their ultimate location, drain into the ethmoidal infundibulum.

The "fronto-ethmoidal cells" in the sense of the terms as used in purely descriptive anatomy, may be derived either from frontal cells

proper, from infundibular or even from the most anterior bullar cells. The frontal sinus likewise may constitute an extension either of a frontal cell proper or of an infundibular cell. This further illustrates the advantages of a nomenclature based on the genetic history of the ethmoidal cells.

DR. O. E. VAN ALYEA (closing): I want to thank Dr. Zimmerman for helping me out on the interpretation of the anatomy in the anterior area. I found these frontal cells that he mentioned opening into the frontal recess in twenty-five cases. No doubt anatomically they are ethmoidal, nevertheless, I, as a clinican, recognize in them frontal sinus characteristics. Infection of those cells gives similar symptoms to infection of the frontal sinus. To me they are still frontals.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

Meeting of Monday, January 9, 1939

THE PRESIDENT, DR. THOMAS W. LEWIS, IN THE CHAIR

Tinnitus: Etiology and Evaluation of the Various Methods of Treatment

DANIEL B. HAYDEN

AND

EDWARD L. CHAINSKI

CHICAGO

(This paper published in full on page 443)

DISCUSSION

DR. EDWARD CHAINSKI: Although tinnitus is generally considered a symptom, it frequently presents itself to us as a disease in itself. A patient may be relieved of the condition responsible for the tinnitus or the pathologic condition producing the tinnitus may cause him little or no concern, yet his life is made miserable by these incessant head noises.

The condition was at one time dismissed with the callous remark to the patient: "Sorry, I cannot do anything more for you." This sort of attitude is no longer being tolerated by either patient or physician. This was forcibly brought home to us by the number of requests from lay people and organizations for reprints of the paper read before one of the Sections of the American Medical Association at the San Francisco meeting.

One gathers from the paper that this is no simple problem. What can we do for these patients? The textbooks on otology do not offer much in the line of treatment. They usually divide all cases into curable or incurable, and unfortunately most of the persistent cases fall into the latter class, and it is to this type of case that we have reference in the paper. The paper does not offer a new or spectacular treatment or tell of any short cuts in the management of the condition, yet I believe it does outline the proper procedure for the care of

patients with tinnitus; and one is often pleasantly surprised at what he can accomplish when he once tries. It is not uncommon after having tried various forms of treatment to have the patient volunteer the information that the intensity of the tinnitus has changed or diminished, and that he is now able to put up with it. Psychologic? Maybe, but we must remember that most patients consult us primarily for relief, and it was said of Osler that many of his successes were due to treating patients with kind words and fresh air.

In reviewing the literature on the subject, one is struck with the difference between the reports in this country and in Europe. The American otologist is concerned more with reporting specific cures. He is inclined to apologize because he has to resort to one treatment after the other without results. In the foreign literature, which is more voluminous, one reads reports of cases under all methods of treatment extending over many years, and finally resorting to radical surgical procedure if warranted. Tonight we are extremely fortunate to be able to hear of a more recent approach to this problem, for although fenestration is done primarily for the relief of deafness, we all know that tinnitus is frequently an early and not uncommonly a most annoying manifestation of otosclerosis. It will be interesting to hear the results obtained by Dr. Lempert and the various discussors.

DR. AUSTIN A. HAYDEN: I have enjoyed hearing this paper tonight. May I add some personal observations on the subject of tinnitus. I have recently been relieved of tinnitus in my left ear. In addition to the recitation and classification of the causes given by Dr. Hayden, I want to add two more. My own, I feel quite sure, was due to excess of uric acid. I say quite sure, because it was not very disturbing but was quite constant and has been for the last six or eight months, and during the general physical examination which I had at St. Joseph's Hospital, it was found that I had a uric acid estimation of 8.1 mgm., which is very high. When that came down to four, I had no more tinnitus in the left ear, and since it came down to 2.6 mgm., I have had no head noises since I left the hospital.

In addition to the consideration that was given to allergy, I would like to venture the opinion that while allergy may work in the middle ear itself, it probably produces tinnitus, and not infrequently through the mechanical obstruction of the eustachian tubes. In that connection I want to mention what I would designate as occupational tinnitus. I have recently made some studies on a group of employees who work in a very high noise level. For thirty min-

utes to an hour after a six-hour day, these men almost without exception have tinnitus in each ear, and it is interesting to note that the tinnitus is about the same pitch as the noise in which they work.

I wish to take exception to what Dr. Hayden has said about the little value of therapeutic treatment. It depends upon what is meant by therapeutics. In my own family within the last three years there has occurred a suicide due to tinnitus. I feel that I was partly to blame, because I gave the exact impression to that individual that this paper may be construed to give. Although his conclusions mentioned suicide, it must be remembered that the individual who is subjected to these explosions, these constant bell ringing and other noises, is a case that needs the most careful handling psychologically, therapeutically and any other way, to give relief.

I was glad the eustachian tube was mentioned, because I believe many cases are relieved by eustachian catheterization with or without the addition of massage, with or without the introduction of any medication into the tube through the catheter.

It was a very strange thing in the industrial examination to which I referred, that while the tinnitus complained of was of very low pitch, corresponding to the noises in the room, the hearing defect present without exception in every case that I examined, was at the uppermost end of the scale, in some cases beginning at 2000, very marked at 4000 and a very large defect at 8000.

Dr. Howard C. Ballenger: Two or three years ago at Northwestern University Medical School, we were treating a series of cases of various kinds of deafness by means of musical tones generated by an electrical apparatus. The primary attempt was to treat the deafness. However, most of these cases had tinnitus. We kept charts and records of the tinnitus and the effect of treatment by sounds and noises. As a rule the tinnitus was not improved. However, in a few cases where we concentrated the sound at about the same frequency as the tinnitus, there seemed to be an improvement in some instances. In such cases, if an excess of stimulation by sound occurred in which the tinnitus was lessened, there was also a decreased hearing acuity for that frequency. This is in accord with the known fact that any excess stimulation of the auditory nerve by noise or concussion will produce partial deafness. The number of cases treated was too small to be other than suggestive, but it might be possible by further experimentation to develop a technique in treating tinnitus; but at the sacrifice of some hearing at that particular frequency treated.

Improvement of Hearing in Otosclerosis: Report of 73 Cases

JULIUS LEMPERT

NEW YORK

(Author's Abstract)

The fenestration for the improvement of hearing in otosclerosis advocated is an endaural, plastic reconstruction of the auditory mechanism for the creation of a new permanent air conduction apparatus. It is based upon definite surgical principles. The degree of improvement in hearing which is obtained as a result of such plastic reconstruction of the auditory mechanism in an otosclerotic ear depends directly upon, and is in direct proportion to the exactitude and perfection with which each major step in this technique is completed, plus the finesse employed in the execution of each and every one of the technique minutiæ.

The fenestration operation herein described has been performed upon 73 cases. These may be divided into two groups, the first consisting of 42 patients who had good bone conduction and were suitable for the operation, and the second consisting of 31 patients whose bone conduction was very poor. These latter cases were operated upon at the request and insistence of the patients in each instance in the hope that they might derive some improvement in hearing, no matter how slight.

Of the first group (those who had good bone conduction), 35 resulted in a high degree of permanent hearing improvement in the operative ear and these patients now enjoy good practical hearing and are socially and economically rehabilitated. In 7 cases in this group the hearing was not improved and remained at its pre-operative level. The hearing was improved in 83 per cent of the cases in this group.

Of the second group (those who had poor bone conduction), 9 cases showed a good hearing improvement, 20 cases remained at the preoperative level and in 2 cases the hearing was further impaired. The hearing was improved in 28 per cent of the cases in this group.

From the above analysis of results in the cases subjected to this operation, it can be readily seen that in properly and carefully selected cases a practical hearing improvement of a high degree can be obtained in a high percentage of cases with this technique.

Tinnitus was either greatly improved or completely disappeared postoperatively in the vast majority of cases.

Though a hearing improvement was obtained and maintained in only 44 cases, the newly created fenestra remained permanently open in 62 of the 73 cases operated upon.

The improvement in hearing obtained following fenestration is one of air conduction. The bone conduction remains unchanged.

The fenestra created in the external semicircular canal can be maintained permanently open by employing the technique which has been described. Of the 73 cases operated upon, the fenestra remained permanently open in 62 cases and closed only in 11, making ultimate closure the exception rather than the rule.

Osteogenesis is not the only cause of closure. Fibrosis finding its way into the fenestra will result in closure and prevent the mobilization of the perilymph and endolymph by air conduction. In the 11 cases in which the fistula closed, a revision of the fistula in the external semicircular canal revealed new bone formation in 9 and fibrosis in 2 cases to be the cause of closure.

Dr. Norton Canfield of Yale has demonstrated experimentally that when creating a defect in bone with the polishing bur employed in this procedure, bone regeneration did not take place. All defects made with sharp instruments and burs were followed by bone regeneration.

The fenestra must be sealed by surgical means. A membrane must be employed to seal the fenestra in order to maintain the hearing improvement obtained. When the fenestra is created in such manner that bone regeneration does not follow, such a fenestra unless surgically sealed with a suitable membrane would still close as a result of fibrosis. The membrane employed to seal the perilymph space must be the thinnest possible and the most vibrating, so as to interfere least with the mobility of the perilymph.

The hearing improvement following fenestration is functional and not psychic. It is always possible to substantiate audiometrically the degree of hearing improvement claimed by the patient.

CONCLUSIONS

1. The Lempert fenestration when carefully and correctly performed will result in a high degree of practical and permanent hearing improvement in properly selected cases of otosclerosis.

2. This operation should not be regarded as just another operative technique added to the list of operative procedures on the temporal bone. This operation blasts a trail for a new and different type of surgery on the temporal bone. It is based upon different surgical principles than any surgery heretofore employed for the relief of suppurative lesions in the temporal bone. The best results from this surgical procedure will be obtained by otologists who have a thorough knowledge and understanding of the surgical principles of plastic reconstructive surgery or by plastic surgeons who have a thorough knowledge and understanding of the anatomy of the temporal bone.

3. Owing to the delicacy and precision of the surgery required to alleviate deafness in otosclerosis, and owing to the difficulty of always definitely determining in advance the suitability of a particular case to this surgery, surgery for the relief of deafness will result in a percentage of failures varying only with the otologists' diagnostic ability and surgical skill.

4. In fairness to the already successful development of the surgery for otosclerosis and for the protection of its still brighter future, no otologist, no matter how skillful a surgeon he may be, should attempt this particular operation without receiving special training in this type of surgery under supervision and guidance.

DISCUSSION

DR. JEROME STRAUSS: It is with a thrill of satisfaction, and a distinct sense of unusual privilege, that I make brief comment on Dr. Lempert and on his work. I have met our guest in New York and have talked with him at great length; I have stood behind him at the operating table, and I have chatted informally with a representative number of his operated patients. To anyone who has done these three things there can be but one possible conclusion—that Julius Lempert is a unique surgical genius and anatomic master of the temporal bone.

A few years ago a delegation of our members returned from a meeting of the American Academy in New York, and reported having seen an endaural surgical approach to the mastoid for acute and chronic disease. One got the impression that the incision was really a very clever manipulation, but that it was considered more or less as a surgical stunt, designed primarily for cosmetic end results. These men, returning to Chicago, spread somewhat the opinions of prominent Eastern otologists at the time regarding both the meatal

incision and its originator; in short, both were something to talk about—let us say with arched eyebrows and an index finger to the nose.

Two years passed, and it would seem that the man who had been demonstrating this incision was now using it primarily only as a stepping stone to bigger and greater things within the temporal bone beneath. From the completely changed atmosphere toward him and his work in the east, it is now manifest that his study and close application to the problems of the temporal bone are beginning to bear fruit, and no one who visits Dr. Lempert, whether for a day or a month, fails to be impressed with the man himself and the work he is doing. Even among his most antagonistic colleagues, who may at one time have questioned his impulses and medical character, he is now hailed as a surgical genius who has made a real contribution to temporal bone surgery, and he has at present among his friends and sponsors some of the best known names in American otology. Indeed this complete reversal of opinion in so short a time reads to those familiar with the details, as a truth that is stranger than fiction.

As to the man himself, I saw Dr. Lempert as an indefatigable worker and an artist. The artist in him takes expression in the collection of many canvases and heterogenous objects of art. As a worker he appears to be tireless in a busy office practice throughout the morning, and performs these patience-taking, time-consuming operations in the afternoon. He finishes his day working on the cadaver until all hours of the night, either for himself or with men who are taking private courses in this work under him.

The operation for improvement of hearing as done by Dr. Lempert is probably the most difficult, complex and lengthy procedure in any field of modern surgery, and his genius is well demonstrated by his infinite capacity for taking pains in such stages of the operation as the cautious fenestration of the semicircular canal, the slow delicate dissection of the tympano-cutaneous flap, and the careful preparation of its bed. The operation I was privileged to witness lasted four and a half hours, with no wasted motion and no time out.

Dr. Lempert was exceptionally candid and hospitable during his office hours, and placed all his patients who were making post-operative visits at the unrestricted disposal of his medical visitors. The oldest case seen had been operated eleven months previously, the newest on this particular day, three weeks. The only one of the group unsatisfactory to Dr. Lempert was this most recent case, who responded to the fistula test somewhat sluggishly in comparison to

the others who reacted with a terrifyingly active nystagmus. Two of the patients seen on this day volunteered the information that they had been spared the loss of self-sustaining employment by the improvement in their hearing.

How long the improvement will last is still, of course, an open question, but there is no longer any doubt Dr. Lempert's method is relatively more permanent in result when compared with any previous attempt for permanent artificial fenestration. He himself made no extravagant claims, and beyond expressing his opinion that if the fistula will last from six to eight weeks, he believes it to be permanent, he is content to let his cases speak for themselves.

There is but one profound criticism to be offered in connection with this magnificient technical procedure—providing, of course, that the fistulæ remain permanent—and that is its unusual complexity. Granted that time will prove the successful outcome of a large percentage of these cases, this serious drawback still presents itself. For, despite the fact that hundreds of thousands of the deafened might clamor for relief through this operation, the sad truth is that the general rank and file of otologists will never be able to do this super-specialized work in the way that Dr. Lempert does it, and the penalties for a misstep are unfortunately very great.

Dr. John R. Lindsay: I have had the privilege of visiting Dr. Lempert and spending some time with him. I have watched him perform the various steps of the operation on several occasions and have had the opportunity of examining patients who had been operated on at various times, some as long as nine months previously. I was impressed with the fact that a special degree of preparation and training is necessary in this most meticulous type of surgery. I was also impressed when I examined the patients who had previously been operated on. There is no doubt that the fenestra has remained open, and there is no question about the marked degree of hearing improvement in patients who had been operated on as long as nine months previously.

Some interesting points have been brought out, I believe, by Dr. Lempert's work. One is that the most favorable cases for operation are those with fixation or ankylosis of the stapes, with at the same time a free round window niche and membrane and good bone conduction. These are the conditions which also give best results with bone conduction hearing aids. We must admit, however, that at the present time there is much objection or diffidence on the part of many people to wearing hearing aids, and it is true that in some

occupations people lose their employment when the hearing is reduced so as to require wearing a hearing aid. Regardless of what the situation may be in the future, this is the condition today. Good results have apparently been obtained only in those cases where the middle ear cavity has been completely sealed off from the external ear and mastoid cavity. This is in agreement with physiologic facts that have been demonstrated experimentally. It may be that the size of the remaining middle ear cavity is a factor in the result to be obtained. The reason why the fenestra has remained open in Dr. Lempert's operation, while those of previous workers have closed, does not as yet seem to be entirely established. The suggestion that the laying of Shrapnell's membrane over the fistula may be responsible for keeping it open, does not seem to be a satisfactory explanation, since the inner surface of the membrane consists of epithelium and the lateral process of the malleus lies at its inferior margin. The experimental work that has been presented here seems to indicate that the polishing bur may be an important fact in preventing osteogenesis. It would seem that a longer period of time must be allowed to elapse in order to prove this point.

I greatly appreciated the courtesy Dr. Lempert showed me in New York and the complete freedom with which he allowed me to examine his patients, discuss various points, and observe the technique of the operation.

DR. SAMUEL J. PEARLMAN: I think it must be quite obvious that we have listened tonight to a most unusual presentation. Everyone knows the hopeless attitude the profession has had about the treatment of otosclerosis. I think I may say without fear of contradiction that no place in the United States, or in the world for that matter, is there an individual who has brought this operation to a higher plane than our essayist. There are a few individuals in Europe who have done this work, one who does a two- or three-step operation requiring several months; another, a man honored in otology, has done the operation over many years, and on occasion has abandoned it. He uses gold leaf for covering the fenestrum which, in my opinion, cannot compare with Dr. Lempert's flap of membrana tympani. This seems to be a technically perfect operation conceived by a person with a high degree of surgical imagination. Here at one step, all the necessities for a proper result are obtained with the use of a viable flap to cover the opening in the labyrinth and made of the thinnest conceivable living tissue.

Seeing these figures, and knowing that Dr. Lempert has been observed for months by men—some of them exceedingly critical—

we will have to say that a great step forward in the treatment of this disease has been made. I would further say that even though the operation is of highest technical difficulty, it should, after practice, be easy in the hands of the more skillful; I would be disappointed if it were shown or thought that only one man could ever do it. I would like to feel that as time goes on there will be otologists of sufficient skill and proper training under careful supervision all over the country who will be able to do this operation. If numbers of men can do it—difficult though it is—following the injunctions laid down by the person who has developed the procedure, definite and important progress will have been made in the treatment of what is the commonest cause of severe deafness in the young adult.

Dr. George E. Shambaugh, Jr.: The Society is fortunate indeed to have such a distinguished otologic surgeon present such an important contribution to otologic surgery. For the first time, the Lempert operation offers a reasonable hope for restoring practical hearing to patients with otosclerosis by a single-stage surgical procedure.

Ten years ago Dr. Lempert introduced the endaural extracartilaginous ant-auricular incision for the simple and radical mastoidectomy. The advantages of the endaural approach for the radical mastoid operation are so evident to anyone who has used it, that I venture to predict that in another ten years the end-aural route will be the accepted procedure for radical mastoidectomies. I have done four radical mastoids endaurally, and will never again use the postaural incision for this procedure. The advantages of the endaural approach for the simple mastoidectomy are less evident and it will be slower in gaining general acceptance, but I believe that eventually the Lempert endaural approach may replace the postaural incision for all mastoid surgery.

Two years ago Dr. Lempert introduced the carotid artery approach for the exenteration of the petrous apex. For cases of closed empyema of the apex without an accessible fistula that can be followed, or cases requiring evisceration of the apex, the Lempert operation is the best approach. I have had occasion to use it. Four months ago a child, aged 7, was brought to me with beginning meningitis, a temperature of 104 degrees, an early stiff neck, 1180 cells in the spinal fluid, ten days after a simple mastoidectomy for a staphylococcus otitis media. Because of the history of intermittent eye pain for a week, and because the x-ray showed a coalescent petrositis, immediate operation on the petrous pyramid was advised.

Skeletonization of the basal labyrinthine portion of the pyramid failed to reveal any suppurative focus or a cell tract leading to the apex. The Lempert technique was then employed to enter the petrous apex, an abscess cavity containing several cubic centimeters of pus being evacuated. Recovery was uninterrupted and complete.

With regard to Dr. Lempert's presentation tonight, his results speak for themselves.

I have employed the Lempert operation in seven cases of otosclerosis during the past six months. The first patient was operated on July 13, the poorer hearing ear being used. The various steps of the procedure were successfully accomplished with improved hearing, which five weeks after operation was sufficient so that he heard definitely better without his hearing aid than he had been able to hear with it before the operation. The fistula then began to close and the fistula test was gone seven weeks after operation, with partial loss of the improved hearing. The operated ear, however, continues to be the better hearing ear.

The second patient was operated on July 20. The membranous flap was created successfully and the fistula into the horizontal canal was made, but the membranous labyrinth was ruptured accidently in making the fistula. There was an immediate tremendous improvement in hearing that lasted two days, after which the hearing became very poor. There was no fistula test until two weeks after the operation, when the fistula test returned indicating regeneration or repair of the membranous labyrinth. The hearing gradually improved to where the operated ear was definitely the better hearing ear. The fistula remains open. The third patient developed a serous labyrinthitis the day after operation with loss of vestibular function, but the hearing remained about the same. The fourth patient obtained a temporary improvement but the fistula test had begun to diminish six weeks after operation with loss of the slight improvement. She then developed a mild serous labyrinthitis with further loss, which is beginning to improve but is still below the preoperative level.

The fifth case was carried out successfully and the fistula has remained open, but no improvement in hearing occurred either on the table or subsequently. Here, again, the membranous labyrinth was ruptured but repair occurred, and there is now a very active fistula test. The explanation of the failure to obtain improved hearing is possibly an occlusion of the round window along with the oval window.

The sixth and seventh cases, done two months ago, each obtained a good temporary result, especially the last case, who experienced an extraordinary improvement in hearing. In both these patients, however, the fistula was enlarged with a sharp curette, a procedure that we now know stimulates osteogenesis, and both fistulas have closed.

As you can see, my results have been meagre. Although the essential steps of the operation have been successfully accomplished, in each case, and in no case was the flap torn or the incus dislocated, the meagre results are due not to the operation but to the operator. Of especial interest is the fact that the membranous labyrinth can be ruptured without destroying the function of the horizontal canal or the hearing. Secondly, even when the fistula closes there may remain a definite improvement in hearing so that the operation on the previously worse ear makes it now the best.

In closing, I think that Dr. Lempert must be commended for the way he has carried out this work on the highest possible ethical plane. He withholds nothing, he emphasizes his mistakes and his failures along with his successes, and his only interest has been in seeing that this work succeeds and progresses, not only in his own hands but in the hands of others. He has spent an extraordinary amount of his time, patience and energy instructing others in how to do this most difficult and delicate and most meticulously exacting of all surgical procedures. His purpose in emphasizing that no one should attempt this work who has not received special instruction is purely to protect the future of this work from the disrepute that will occur if the operation is attempted without the necessary preparation.

Dr. Joseph C. Beck: I rise particularly to voice my appreciation, and for most of you I am sure, for the work presented here tonight, but I rise also personally because I have to take my finger from my nose, as Dr. Strauss stated, and put my hands to my head in wonderment, so to speak. Ten years ago I visited Dr. Lempert because I heard that there was a wonder boy in New York, and I came away not pleased. He impressed me rather poorly, except for the very kind way he received me. But the work did not satisfy me because of the orthodoxy I had enjoyed in my training, and his demonstration was quite a departure. So I did not take to anything he said, personally or in print. But I surely began to change my mind when that galaxy of Chicagoans, also referred to by Dr. Strauss, my own associate, Dr. Guttmann, among them, returned and described

this man's work. However, I still did not go so much for it, but I think we all appreciate now that this was a mistake on my part.

In Sweden, in 1927, I visited Upsala and Stockholm and saw Bárány and Holmgren do this operation for fenestration of the horizontal semicircular canal. In 1931, I performed an operation for the fenestration of the horizontal canal at Michael Reese Hospital, on a young woman with otosclerosis, and had a most remarkable result. After she was returned to bed from the operating room, she said the voices she heard were too loud; she had not heard anything like that for a long time. Also she lost the tinnitus from which she was suffering, and I was very much pleased. My operation was done with the bur, but the Swedish gentlemen used sharp chisels and curettes. The bur had not up to that time been used universally in general mastoid surgery in this country. I have employed it over a period of years. But using it in the way Dr. Lempert has described, is something we always tried to avoid because of the hazard of burning the bone and causing subsequent sequestration.

I would like to ask Dr. Lempert whether the age of the patient would be considered as having anything to do with osteogenesis, and whether that would be considered in connection with the success of the operation. We know that reparative osteogenesis is much greater in younger individuals. I would also like to know if he considers that the inner lining of Shrapnell's membrane and the proper tympanic membrane, which are covered with epithelium, play a part, or does he consider that the epithelium becomes transformed into fibrous tissue and becomes adherent over the made fenestrum. That is one factor which it occurred to me might be responsible for the nonclosure of the opening.

Brunning referred to operations for otosclerosis in the Zeitschrift fur Hals. Nasen u. Ohrenh., and gave a review of the literature. Lempert has been on the map for some time, but not a word was mentioned by him about Lempert; everybody else was mentioned. I feel that this is unjust. Then I see that the English have also reported on this work in the Journal of Laryngology, and in particular Howard is reported to have operated by fenestration of the canal on several occasions. They said that closure is universal and the operation is a failure. Brunning brings out an operation on the superior semicircular canal which he says is much easier in keeping the fenestra open.

Again I wish to express my appreciation to Dr. Lempert for this presentation.

Dr. Alfred Lewy: We must realize, after listening to Dr. Lempert, that we may be on the eve of an epoch-making event in otology. It sounds like the realization of the otologist's dream. But we have to be careful. Medical history is replete with operations that were successful and brought great credit to their originator for a time and then went into the discard. In 1876, Kessel began removal of the malleus, incus and membrana tympani for suppurative deafness, followed by Lucae in 1885 for nonsuppurative deafness. Many other operations have been devised, most of which I am omitting, but I want to mention removal of the stapes, which was done by Blake of Boston, and Jack of Boston in 1892. In 1894, Jack reported his results on 60 cases as discouraging. The operation of fenestration or fistulization of the external wall of the labyrinth was done by Floderus in 1899. After dissections, study of microscopic serial sections and animal experimentation, he suggested what he called "sound fistulas" in the labyrinthine wall near the oval window or in the horizontal semicircular canal. This preceded Bárány's work or that of Sourdille and Holmgren. It is perfectly possible that the failure these men encountered and their discouragement was due to faulty technique.

Dr. Lempert's work was presented before the American Otological Society by Dr. Kopetzky on 42 cases. The discussion was opened by Dr. Wishart of Toronto, who visited Dr. Lempert twice, saw him operate, and examined some of his cases. He spoke in the highest terms of Dr. Lempert's exquisite operative skill, but expressed himself as dissatisfied (through no fault of Dr. Lempert's) with his own investigation of the hearing before and after operation. He felt they were inadequate to form any opinion as to the end-result of the operation. Time enough had not elapsed. Since then more time has elapsed and Dr. Lempert has some excellent reports to make. That same opinion seemed to be the consensus of other members of the society in the discussion that followed. They believed that adequate examinations of the patients before operation had not been made by a sufficient number of men, and the same applied to patients after operation. That, of course, will be corrected in time.

With the history of surgical procedures in medicine before us, I am glad to see that Dr. Lempert in his closing paragraphs was very careful to say something along the same lines, to warn us against too much enthusiasm until enough time had elapsed for mature judgment; also to warn us that this operation must not be undertaken without adequate surgical preparation on the part of the operator.

In closing, Dr. Wishart remarked that we never would know until a Scotch doctor from Missouri made observations on a Scotch patient from the same state. That, of course, is how scientific medicine advances.

As we look into the canal and see Shrapnell's membrane, it does not appear to be wide enough to cover a 6 mm. trough. Perhaps if dissected free there is more of it. I have not done the operation and I do not know the answer.

I want to thank Dr. Lempert for coming here and giving us this wonderful presentation.

Dr. Samuel Salinger: If one were to cite all the failures of the various operations for otosclerosis which have been tried and discarded since 1876, he could take up the entire evening. Dr. Lewy touched on a few of them, but it all comes to the same point, that no one was ever able to maintain this fenestra in the canal or promontory, or wherever they put it, for a sufficient time to insure permanence. Wittmaack's operation on the superior canal went further than has been mentioned; he excised a portion of the dura to allow the arachnoid to come in contact with the membranous canal. That procedure improved the tinnitus but did nothing for the hearing. I, too, had the pleasure of seeing Dr. Lempert do his operation, and I must admit that I was very much impressed with the details of the procedure as well as the great technical skill displayed by the operator. In fact, I made the statement to Dr. Lempert that there were not more than a dozen or fifteen men in the country who were capable of carrying out the technique as he did. As for the duration of the procedure, one must bear in mind that operations lasting more than fours hours are not uncommon in brain and spinal cord surgery. Surely, if the neurosurgeon can stand for six hours over a brain tumor, there is no reason why the otologist cannot do likewise if the procedure is justified. However, this operation will fall into discredit if it is done by men inadequately trained. Old men cannot learn it. It is for the young men to begin on, who have sufficient time and plenty of stamina. I believe there are enough men who can learn to perform this operation as Lempert does it, so that eventually sufficient evidence will be accumulated as to its ultimate value.

Dr. T. C. Galloway: I would like to make up for Dr. Pearlman's modesty. We have been using the endaural surgical approach in mastoid operations and have been impressed with its value, practicability and reasonableness.

Probably everything said about the difficulties of the fenestration operation is correct. It must not be done without a good background of experience, anatomic knowledge and skill. For that reason, at County Hospital, we have delegated to Dr. Pearlman the tryout of the fenestration operation, and he has done a number of cases.

The patients have gotten along well; the results are not old enough to determine the final results, but there have been remarkable improvements in hearing, and I wish Dr. Pearlman had brought down a case seen this morning—beautifully healed, a smooth flap over the fenestration, the patient in excellent condition, and with nearly normal hearing. Dr. Pearlman has evidently been able to master the procedure, and I think there are other men who can follow in Dr. Lempert's footsteps.

DR. JULIUS LEMPERT (closing): I would like to tell Dr. Shambaugh why the operated ear in five of his seven cases became the better ear in spite of the fact that the fistula closed. Even where a fistula closes due either to new bone regeneration or fibrosis, the possibility exists that the new wall covering the membranous labyrinth is not as thick and as firm as the original bony labyrinthine capsule. This results in a certain amount of mobility of the perilymph which may account for the five or ten decibel increase in hearing which results in these cases.

In reply to Dr. Beck, with regard to the effect of heat produced by the bur, the polishing bur is employed in such a manner that at no time is there real heat produced. I never make more than two to three strokes at a time with the bur, and then stop, employing very little pressure with each stroke. With regard to the effect that age had upon bone regeneration, so far as I observed it, age did not seem to make any difference. In nine cases there was new bone formation, in two cases fibrosis. The age of these patients varied between 19 and 45. Most of the entire group were young, a few were above the age of 55; the ages ranged from 17 to 55. Age is no contraindication to operation provided there is a perfect state of health.

Whether Shrapnell's membrane has anything to do with prevention of osteogenesis, I do not know. But I do know that it is important to seal the fenestra with the thinnest possible vibrating membrane in order to maintain the necessary mobility of the perilymph and endolymph; and that this membrane should not be an independent membrane but rather a part of a larger membrane with a good blood supply. It is for this reason that I seal the tympanic

cavity with the membrane tensa and extend the membrane flaccida backward to cover the fenestra in the external semicircular canal.

Dr. Lewy stated that Dr. Wishart said he was unable to make a proper analysis of the audiometric readings in the cases reported. This was due to the fact that Dr. Kopetzky did not send the paper to him in time to permit such analysis. Why Dr. Wishart did not receive the paper ten days prior to the presentation by Dr. Kopetzky, as promised, is something that I cannot go into now, for this is neither the time nor the place for such a discussion. Every patient who was operated on had not only an adequate examination by myself, pre- and postoperatively, but by other most prominent otologists in the city. Every patient who will submit to this operation has made the rounds and consulted most of the reputable otologists in his community, and very often consulted otologists in different parts of the country and the world. The patient who submits to this operation does so only after all other available methods of treatment have failed. Furthermore, it is difficult to conceive how any patient who can hear will seek and submit to surgery for the relief of deafness.

My patients are all private patients. Every one of them always stood ready to be examined and was examined by some of the most prominent men in the country. Any otologist of standing who asks to see my material is always welcome, not only to see the patients, but to witness operations and examine the patients before and after operation. Many otologists have seen these patients regularly before and after operation, so there never can be any question about the hearing of these patients.

Dr. Wishart has been doing cadaver work himself for many months, and I believe has already started to do otosclerosis operations. In answer to Dr. Lewy's question as to how Shrapnell's membrane can be made to cover the fenestra in the semicircular canal—by liberating the tympanic membrane from the sulcus tympanicus, leaving it attached only to the anterior and inferior walls of the sulcus. The tympanic membrane is extended in a posterior direction until it reaches far enough to cover the external semicircular canal.

Dr. Salinger and Dr. Galloway both spoke about the mastering of this operation. It is not a one-man operation. Any otologist with good understanding of the anatomy and surgery of the temporal bone, plus a keen understanding of the surgical principles of plastic surgery, after dissecting a dozen cadavers under the guidance of someone who has done this operation, will readily master this tech-

nique. Many otologists will be able to do it. It is time-consuming only in the beginning, when the surgeon is not quite familiar with the technique. It has taken me from three to six hours. One man whom I trained took eight hours to do his first case and five hours for the second. He has done six operations, five absolutely successful. He was a well-trained man from the Mayo Clinic. It is not necessary to take a course, it is just necessary to apply oneself, see it done a number of times by one who knows the technique. The correct execution of the technique minutiæ, even such small detail as the placing of the paraffin mesh, correctly, may mean complete defeat of the whole operation if not done properly. These things are most important to the end result, but can be learned very easily. One way is through bitter experience by oneself, and the other by being guided by someone who has gone through this bitter experience. The operation can be done and will be done by anybody who understands the anatomy of the temporal bone.

Finally, I wish to thank the President and the Society for being permitted to bring this work before you, and I assure you that my institution, my office, my patients, myself and my cadaver material are at the disposal of every otologist from Chicago who is interested in this work and in seeing the cases at any time.

Harris N. Vail

HARRIS HOLMES VAIL

1892-1939

Dr. Harris Holmes Vail, of Cincinnati, died April 10, 1939, in that city.

Dr. Vail graduated from Yale University in 1912 and from Harvard Medical School with Cum Laude in 1917. He served as an interne at Peter Bent Brigham Hospital, and then as a physician at the Massachusetts Eye and Ear Infirmary.

He was a member of the Cincinnati Academy of Medicine, Ohio State Medical Society and the A. M. A. He was also a member of the American Otological Society, American Laryngological Association and the American Academy of Ophthalmology and Otolaryngology. He participated actively in the local and national societies to which he belonged. He was a member of the staff of Christ Hospital, Good Samaritan Hospital and the Cincinnati General Hospital. During the World War he served in the Naval Medical Corps.

Dr. Vail was generally beloved and respected for his kindly nature and his sincere interest in his work and his patients. He approached his medical problems with an intelligent insight and honesty which earned universal respect for his views. His most significant work was on the infections of the sphenoid sinus and their effects upon the vidian nerve.

He is survived by his widow, Adelaide Brush Vail, three children, Virginia, Charlotte and Charles. Dr. D. T. Vail of Cincinnati is his only surviving brother.

A partial list of his publications is appended.

PUBLICATIONS OF DR. HARRIS HOLMES VAIL

1. Studies by the Bárány Rotation and Caloric Tests of Tumors of the Nervous Acusticus. The Laryngoscope, St. Louis, Aug., 1920.

2. Anatomical Studies of Dorello's Canal. The Laryngoscope, Aug., 1922.

3. Eye, Ear, Nose and Throat Symptoms of Encephalitis Lethargica; Report of Two Cases. Cin. Journal of Medicine, May, 1923.

4. A Case of Ivory-like Osteoma Originating from the Os Planum and Invading the Orbit. The Laryngoscope, June, 1923.

5. A Case of Epidermoid Carcinoma of the Epiglottis. Cin. Journal of Medicine, Nov., 1924.

6. Some Considerations of So-Called Nasal Catarrh. Cin. Journal of Medicine, Nov., 1924.

7. Vidian Neuralgia from Disease of the Sphenoidal Sinus. Archives of Surgery, 18:1247-1255, April, 1929.

8. Traumatic Conditions of the Ear in Workers in an Atmosphere of Compressed Air. Archives of Otolaryngology, 10:113-126, Aug., 1929.

9. Rhinological Problems of Interest in the Practice of Medicine. Cin. Journal of Medicine, Jan., 1930.

10. Retrobulbar Optic Neuritis Originating in the Nasal Sinuses: A New Method of Demonstrating the Relation Between the Sphenoid Sinus and the Optic Nerve. Archives of Otolaryngology, 13:846-863, June, 1931.

11. Practical Consideration of Sphenoidal Sinus Infection. Ohio State Medical Journal, July, 1933.

12. Vidian Neuralgia. Annals of Otology, Rhinology and Laryngology, 41:837, Sept., 1932.

13. Vidian Neuralgia with Special Reference to the Eye and Orbital Pain in Suppuration of the Petrous Apex. Archives of Otolaryngology, 17:212-221, Feb., 1933.

14. Schwannoma of the Larynx. Annals of Otology, Rhinology and Laryngology, 45:476, June, 1933.

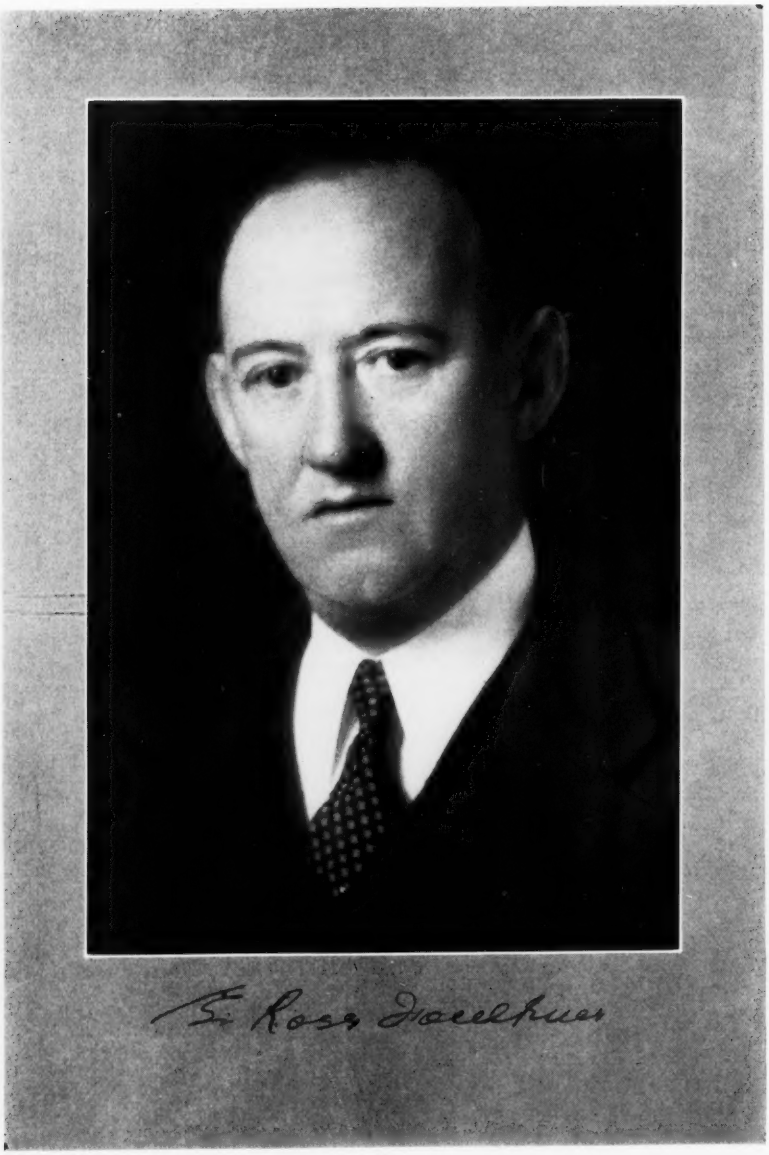
15. Treatment of Hay Fever by Nasal Injections of Alcohol. Archives of Otolaryngology, 18:651-655, Nov., 1933.

16. Pathways of Reflex Pain in Vidian Neuralgia. Archives of Otolaryngology, 21:277-284, March, 1935.

17. Headache—From the Standpoint of the Otolaryngologist. Transactions of the Amer. Acad. of Ophthalmology & Otolaryngology, 1935.

18. Neuralgia of the Face and Head. Cin. Journal of Medicine, July, 1936.

19. Neuralgia of the Head and Face Distinct from Trigeminal Neuralgia. Practitioners Library of Medicine and Surgery, Vol. 40, Chapter 45, published in 1937 by Appleton, and Edited by Professors George Blumer, Arthur M. Yudkin and Paul B. MacCready.


E. Ross Doelker

E. ROSS FAULKNER, M.D.

1876-1939

Dr. E. Ross Faulkner, Surgeon Director at the Manhattan Eye, Ear and Throat Hospital for the past 16 years, died suddenly at his home on May 29, 1939, of coronary thrombosis at the age of 63.

Dr. Faulkner was born in Glenholme, Nova Scotia, on January 28, 1876, the son of Captain Thomas Faulkner and Arabella Morrison Faulkner, and was one of seven children.

He attended Pictou Academy and then received his A.B. degree from Dalhousie University in 1897 and his M.D.C.M. degree from the same school in 1901. He interned in a Halifax hospital and then began the practice of general medicine in Rose Bay, Nova Scotia. He decided to give up general practice and study general surgery, which took him to the University College Medical School and St. Thomas' Hospital Medical School in London, England. His post graduate study culminated in his becoming a licentiate of the Royal College of Surgeons in 1908.

In the autumn of that year he came to New York to establish a home and a practice. In 1915 he married Miss Julia Whiton Little of New York. No children were born to this happy union.

For two years he taught anatomy and surgical pathology at New York University Medical School and later taught the same subjects at Cornell University School of Medicine.

Subsequently he specialized in Otolaryngology and became associated with Manhattan Eye, Ear and Throat Hospital in 1910. He was associated with this hospital until the time of death. In 1923 he became one of the hospital's surgeon directors. During the World War he volunteered for military service and was assigned to a Draft Board. For several years he was Professor of Ear, Nose and Throat Surgery at New York Polyclinic Medical School.

He was one of the country's outstanding otolaryngologists and an authority on the diseases and surgery of the sinuses; the author of many articles, as well as a contributor to Jackson and Coates Textbook on "Diseases of Ear, Nose and Throat," and edited a monograph on "Diseases of the Larynx."

Dr. Faulkner was a member of the American Laryngological Association, The New York Laryngological Society, New York

Academy of Medicine, American Medical Association and the New York State and County Medical Societies. He was a member of the Council of the American Laryngological Association at the time of his death.

Dr. Faulkner was a man of great personal charm and was widely recognized as occupying a high place in his chosen profession. He will be greatly missed by his fellow practitioners, as well as his many personal friends, and his loss will be keenly felt by all those who came under his sphere of influence.

WILLIAM F. HUBBLE, M.D.

PUBLICATIONS OF DR. E. ROSS FAULKNER

1. Two Cases of Sinus Thrombosis which Presented Unusual Difficulties in Diagnosis. Annals of Otology, Rhinology and Laryngology, 1918.

2. Ozena, Secondary to Chronic Sinusitis—Operation and Recovery. Ibid., 1918.

3. Tic Douloureux with Special Reference to Treatment by Alcoholic Injections. Laryngoscope, 1919.

4. The Intranasal Sinus Operation with Special Instruments. Laryngoscope, 1920.

5. New Method of Exposing the Pituitary—with Report of a Case. Laryngoscope, 1920.

6. Treatment of Intranasal Suppuration. New York State J. M., 1921.

7. The Relation of Certain Ocular and Cerebral Conditions to Infections of Nose and Throat. New York State J. M., 1922.

8. The Relation of Sinusitis to Arthritis Deformans. Trans. Am. Laryngol. Assoc., 1923.

9. The Problem of Focal Infection in the Nasal Sinuses. Laryngoscope, 1925.

10. Papilledema from the Viewpoint of the Rhinologist. Laryngoscope, 1926.

11. Edited a Book on Malignant Diseases of Larynx, written by John E. MacKenty, M.D., in Sept. 1934.

12. Diagnosis in Unusual Cases of Sinus Disease. Laryngoscope, 39:744-746 (Nov.), 1929.

13. Radical Operation of Antrum. Laryngoscope, 40:10-13 (Jan.), 1930.

14. External Operation on Ethmoids. Laryngoscope, 40:646-647 (Sept.), 1930.

15. Rhinological Aspects of Cavernous Sinus Thrombosis. Surg., Gynec. & Obst., 52:474-478 (Feb.), 1931.

16. Place of Otorhinolaryngologist in Treatment of Eye Diseases. N. Y. State Journal of Medicine, 311:1386-1389 (Nov.), 1931.

17. End Results of Sinus Surgery. Laryngoscope, 42:614-620 (Aug.), 1932.

18. Atresia of Pharynx and Other Plastic Operations Developed by Dr. MacKenty. Laryngoscope, 43:103-105 (Feb.), 1933.

19. Present Status of Radical Sinus Surgery. Laryngoscope, 45:782-785 (Oct.), 1935.

20. Conservative and Radical Surgical Methods of Treatment. Trans. Am. Laryng., Rhin. and Otol. Soc., 41:88-97, 1935.

21. Problems in Diagnosis and Treatment of Hyperplastic Sinusitis and Allergy. Annals of Otology, Rhinology and Laryngology, 47:144-152 (March), 1938.

Abstracts of Current Articles.

NOSE

Acute Osteomyelitis of the Superior Maxilla in Children: Report of a Case.

Lacy, M. Eugene, and Engel, Lawrence P. (Kansas City, Mo.), Arch. Otolaryng., 29:417 (Mar.), 1939.

One case of osteomyelitis of the superior maxilla is reported along with a rather thorough review of the literature on this subject. The author feels in this case that the etiology of osteomyelitis was hematogenous.

In considering treatment, early operative intervention is advocated with establishment of drainage primarily through the mouth and secondarily through the antrum. Whenever possible, drainage through the skin of the cheek should be avoided because of deformity.

The authors feel that maxillary sinusitis is a complication of osteomyelitis of the maxilla rather than the cause of it and feel that the prognosis in these cases is very good.

TOBEY.

PHARYNX

Parapharyngeal Hemorrhage: Diagnosis and Treatment.

White, Francis W., and Huber, Louis (New York), Arch. Otolaryng., 29:1 (Jan.), 1939.

Six cases are reported by the authors in which the common carotid was ligated twice, the external carotid twice, the ascending pharyngeal artery once, and involvement of the jugular vein once. Horner's syndrome occurred once and partial facial paralysis occurred in one case. In all of the cases pharyngeal symptoms were found either early or late. The discussion follows under several heads, such as anatomic and diagnostic conditions of the region involved by the infection and hemorrhage; also involvement of the nerves in the pharyngeal maxillary space, and, finally, the problems of treatment of these cases. Many cases of pharyngomaxillary infection are found to get well with conservative treatment. However, free active hemorrhage from the throat with or without apparent infection require bold external operative measures. Further aural

hemorrhage is associated with pharyngomaxillary infection. Immediate ligation of the common carotid artery is the treatment of choice. In conclusion, hemorrhages of the throat associated with trismus, nerve involvement, lateral pharyngeal swelling, sepsis and swelling and tenderness of the corresponding side of the neck indicate infection of the pharyngomaxillary fossa.

TOBEY.

The Pathology of Nasopharyngeal Tumors.

Cappell, D. F. (Dundee), J. Laryng. and Otol., 53:558 (Sept.), 1938.

The author presents a study of 122 tumors of the nasopharynx, pharynx and tonsils. Emphasis is placed on the lympho-epithelioma group, which made up ten per cent of the cases.

The lympho-epitheliomata are divided histologically into: (a) the type corresponding to the description of Regaud, (b) the type described by Schmincke, and (c) transitional-celled carcinoma.

It is concluded that, when dealing with nasopharyngeal carcinomata, biopsy is harmless but further surgery is useless, irradiation being the treatment of choice.

An interesting case of rhabdomyoma of the palate is presented in detail.

DEAN, JR.

LARYNX

Pneumopericardium and Pneumomediastinum in Cases of Acute Obstructive Laryngitis.

Graebner, Herbert (New York), Arch. Otolaryng., 29:446 (Mar.), 1939.

Of twenty tracheotomized patients, four were shown to have air in the mediastinal structures; also, one case is included in which tracheotomy was not performed. Pneumopericardium and pneumomediastinum may occur at any time in the course of an acute obstructive laryngitis. Their occurrence is directly influenced by obstruction of the upper and lower part of the respiratory tract. The complication was not found to be the result of tracheotomy, for it occurred in one case in which tracheotomy was not performed, and in another case it was present prior to operation. In three cases in which tracheotomy was performed and care was taken to seal the wound against air, the complication did occur. Therefore, it may occur with or without operative procedure.

These complications do not influence materially the course or prognosis of the disease with which they are associated. The mechanism by which spontaneous pneumopericardium is produced is unknown. There are comparatively few reports of pneumopericardium and pneumomediastinum.

However, this author is of the opinion that it occurs quite frequently and that repeated roentgen and physical examinations at short intervals in cases of acute obstructive laryngitis would probably reveal positive examples of these conditions.

TOBEY.

The Larynx in Infancy: A Study of Chronic Stridor.

Heatly, Clyde A. (Rochester, New York), Arch. Otolaryng., 29:90 (Jan.), 1939.

This article is based on a study of twenty cases of chronic stridor in infancy. "Chronic stridor in infancy, like chronic hoarseness, requires a careful direct study of the larynx. In the majority of cases the cause will be found within the larynx itself in the form of a flaccid superior aperture, an overdeveloped flabby epiglottis, palsy of one or both vocal cords, a tumor, web, cyst or hypertrophy of the subglottic tissues or ventricular bands. The tendency to ascribe the respiratory disturbance to extrinsic causes, particularly thymic pressure, solely on the basis of suspicious roentgenologic changes, without direct laryngoscopic examination, is still common enough in pediatric practice to impose on laryngologists the duty of urging consultation on all such problems. With proper diagnosis the possibilities of successful management are excellent enough to permit a favorable outcome in the majority of instances."

TOBEY.

EAR

Studies of Pupils of the Pennsylvania School for the Deaf: I. Auditory Acuity.

Hughson, Walter (Abington, Pa.); Ciocco, Antonio (Baltimore, Md.); Palmer, Carroll (Washington, D. C.), Arch. Otolaryng., 29:403 (Mar.), 1939.

Audiograms for air conduction of 460 pupils and for bone conduction of 454 pupils of the Pennsylvania School for the Deaf are herewith analyzed from a statistical standpoint. Complete lack of response to auditory stimulation is a rare condition. Ninety-five per cent of the 460 children responded to at least one tonal frequency by air conduction in one ear, and 85 per cent, in both ears. In the great majority of these children the loss of hearing is bilateral and

approximately equal in the two ears. Sex is not a significant factor. Since age is not related to the degree of loss, the type of deafness dealt with here does not seem to be progressive.

Fifty-four per cent of the children were said to have been born deaf. The percentage of children who presumably became deaf after birth is highest at one year of age and decreases rapidly at each successive age. No difference in acuity of hearing could be found between the children who were born deaf and those who were reported as having acquired deafness later in infancy or childhood.

The deafness developing after birth has been attributed to numerous pathologic conditions, among which meningitis is the most frequent. This disease was the stated cause for deafness in 17 per cent of the children. Meningitis, together with trauma of the head, measles, otitis media or scarlet fever was given as the cause of deafness for almost one-half the children. There is apparently no association between auditory thresholds and stated causes of deafness. A significant number of the children examined were found to have enough residual hearing either by air or by bone conduction to warrant some rational effort toward therapeutic relief.

TOBEY.

The Petrous Apex of the Temporal Bone and Its Relations.

Mellinger, Wm. J. (Santa Barbara), Arch. Otolaryng., 29:484 (Mar.), 1939.

Individual susceptibilities and anatomic variations are common and must be taken into account in the evaluation of signs and symptoms accompanying infection of the petrous apex.

The temporal bone is unique in its connection with other bones. Provision is made for movement and growth with minimum stress on or shock to the hearing apparatus.

The petrous portion of the temporal bone has two intracranial and one extracranial surface, all of which are in communication with each other through the venous spaces at the mesial end. The cerebral and the cerebellar surfaces are separated by the firm attachment of the tentorium cerebelli along the superior border of the pyramid, except at the mesial end.

The petrous apex is that portion of the pyramid mesial to the labyrinthine capsule. An imaginary line beginning at the notch for the exit of the cochlear duct and ending at the hiatus fallopii would mark its lateral boundary.

In connection with the cerebral surface of the petrous apex, it is important to remember its relation to the temporal lobe, the greater and the lesser superficial petrosal nerves and their foramens and to the foramen for a branch of the middle meningeal artery; its impression for the gasserian ganglion, where the bone is thin and perforated by small openings for the transmission of veins in about 75 per cent of specimens examined; its relation to the foramen lacerum for the internal carotid artery, the pericarotid venous plexus and the carotid sympathetic plexus; its proximity to the foramen spinosum for the middle meningeal artery and vein and a recurrent branch of the sensory portion of the mandibular nerve, which may account for parietal pain, and its relation to the foramen ovale for the mandibular nerve and the venous connections between the cavernous sinus and the pterygoid plexus.

In connection with the cerebellar surface of the petrous apex, it is important to remember its relation to the pons, medulla and the cerebellum, to the abducens nerve, to the internal auditory meatus for the facial and the auditory nerve and the auditory artery and vein, to the hiatus cochlearis for the cochlear duct and an artery and vein and to the groove for the inferior petrosal sinus, and its proximity to the subarcuate fossa for the passage of a small artery and vein and to the jugular bulb, the glossopharyngeal and the spinal accessory nerves.

In connection with the extracranial surface of the petrous apex, its location at the upper end of the carotid sheath must be kept in mind, as well as its relation to the eustachian tube, the groove for the sinus of Englich and the carotid canal. It is perforated for the passage of small veins and is situated close to the pharyngomaxillary space, the lateral pharyngeal recess and the retropharyngeal space.

The abducens nerve is placed in a vulnerable position on account of its intimate relation to venous spaces throughout most of its long course, also on account of its relation to the basi-sphenoid and the petrous apex.

The petrobasilar suture may be fused with the diploic structure of the petrosa continuous with that of the sphenoid and occipital bones. Thus osteomyelitis may extend in either direction.

The process of pneumatization seems to follow the formation of the diploe; therefore, cancellous structure is usually found along with the air spaces. Thus osteomyelitis and osteitis are often present at the same time.

Venous spaces along the internal carotid artery and the petrobasilar suture may, on the roentgenogram, be mistaken for pneumatic cells.

The Gradenigo syndrome is often not complete and therefore should not be alone relied on for diagnosis or therapy.

TOBEY.

Process of Healing in Injuries to Capsule of Labyrinth.

Perlman, H. B. (Chicago), Arch. Otolaryng., 29:287 (Feb.), 1939.

A study of 52 experimental animals examined histologically following injuries to the labyrinth is recorded. The means by which repair takes place are given in detail. The animals were killed from a few hours to twenty-five days following injury. A summary of the healing process is as follows:

"A small perforating injury of the capsule of the labyrinth is sealed by a blood clot after a few hours. Swelling of the adjacent periosteal layer is observed after the first day. The fibrous tissue proliferating from the periosteum then invades the blood clot and replaces it in three days. At the same time a differentiation of the periosteum into osteogenic layer, or cambium, and fibrous layer occurs. By the fifth day apposition of new bone begins against bone chips and the surfaces of the fracture as well as on cortex where the periosteum was injured. By the end of the second week most linear fractures are well filled with bone, along with vascular tissue, which has grown in with it from the periosteum. These blood vessels continue into the depth of the perforation, probably anastomosing with the intralabyrinthine vessels. The new bone and its nutrient vessels remain distinct from the adjacent bone of the capsule. At the end of four weeks excess periosteal callus and to a lesser degree endosteal callus are removed, and the activity of the tissues at the site of injury appears to have reached equilibrium. New bone gradually invades and replaces them without definite evidence of osteoclastic absorption in it or in the periosteal callus. Few fragments or spicules are to be seen in the callus after thirty days. The endochondral and the endosteal layer of the capsule forming the walls of the perforation show no evidence of osteogenic activity. The connective tissue of the endosteum and the connective tissue covering the lamina between the coils and in the spiral ligament respond to injury by laying down a meshwork of delicate fibrous tissue, in part of which ossification may begin by the eighth day. Some of the spiral ligament itself at

the site of injury and for some distance away ossifies. Small localized perforating aseptic injuries to the cochlea of the rabbit do not result in a progressive obliterative process within the labyrinth, such as is produced by a suppurative process within the labyrinthine spaces."

TOBEY.

Observations on the Pathology of Ménière's Syndrome.

Hallpike, C. S. (London), and Cairns, H. (London), J. Laryng. and Otol., 53:625 (Oct.), 1938.

Two cases are reported in which the temporal bones were secured and studied after death following operation for section of the eighth nerve for persistent vertigo.

Both cases showed, in the involved labyrinth, distention of the endolymph system as evidenced by dilatation of the saccule and scala media. Both cases had degeneration of the organ of Corti and one showed degeneration of the maculæ and of the cristæ of the semicircular canals.

Absence of the perisaccular connective tissue in both cases makes the authors feel that this may be of etiological significance, since this connective tissue is thought to be the normal absorption area of the endolymph (Guild).

DEAN, JR.

Chronic Suppurative Lesions of the Petrous Pyramid: Report of Six Cases with Different Pathologic Characteristics.

Lillie, Harold I., and Williams, Henry L. (Rochester, Minn.), Arch. Otolaryng., 29:345 (Feb.), 1939.

Examples of pathology occurring as a result of chronic suppurating processes such as meningitis, cavernous sinus thrombosis, brain abscess and abscess formation with rupture into the nasopharynx are reported in six cases. "Two were cases of encapsulated chronic petrositis without apparent otitis media. While it is not comforting to think of apparently healed mastoiditis or petrositis becoming active again after a period of five or ten years, nevertheless, the conclusion that it can do so seems inescapable.

It is apparent that in certain cases in which there is an obscure clinical syndrome referable to the head, the history of an ancient

aural disease may assume a place of great importance in determining the lesion causing such a syndrome. Such cases as are here presented show the futility of attempting to fit clinical material into didactic or academic pigeonholes, because the cardinal symptoms and signs of petrositis were absent in whole or in part in each of the six cases. If the presence of the serious lesion in the petrous pyramid had not been suspected and exploratory operation had not been instituted before the appearance of complete symptoms and signs, the result might not have been so favorable.

The value of exploratory operations on the temporal bone when the condition is obscure and one feels strong probability of a serious disease has been demonstrated to our satisfaction. This group of cases demonstrates to us that it is necessary to approach the petrous pyramid without preconceived ideas regarding the surgical technique that will be adequate for the control of the disease encountered."

TOBEY.

Thrombosis of the Lateral Sinus: A Study of Fifteen Cases, Including Histologic Examination.

Druss, Joseph G. (New York), Arch. Otolaryng., 29:42 (Jan.), 1939.

This is a presentation of fifteen cases of thrombophlebitis of the sigmoid sinus and jugular bulb with histological studies of the affect on the temporal bones. The paths of infection in the temporal bones and the various sinuses were studied thoroughly microscopically.

Thrombophlebitis of the sigmoid sinus may or may not be localized during life, but is readily found by histologic examination. Infection of the jugular bulb was found in all of the fifteen cases examined. The infection of the bulb usually comes from an infected sigmoid sinus but may extend directly from the tympanic cavity. If this occurs it is known as primary thrombosis of the bulb.

Careful investigation of all the various sinuses is stressed because of the possibility of infection through these pathways to the meninges. Thrombosis of the superior and inferior petrosal sinuses was found not infrequently on histological examination.

The treatment of sinus thrombosis should be ligation of the internal jugular vein, incision and removal of infection within the sinus, and obliteration of the vessel.

TOBEY.

A Fatal Otitic Cerebellar Abscess in a Child Four Months of Age.

Adams, W. Stirk (Birmingham), J. Laryng. & Otol., 53:668 (Oct.), 1938.

This case is of interest because of the rarity of otitic brain abscess in children under one year of age. There were no symptoms or signs to indicate the presence of an intracranial complication of the bilateral acute mastoiditis. At autopsy an abscess was found in the right lateral lobe of the cerebellum.

DEAN, JR.

MISCELLANEOUS

Osteomyelitis of the Skull Complicating Mastoiditis and Frontal Sinusitis: Report of Two Cases.

Hirst, Otto C. (Philadelphia), Arch. Otolaryng., 29:24 (Jan.), 1939.

The possibility of osteomyelitis should be borne in mind in each case in which a simple mastoidectomy is to be done and in those cases in which the naso-frontal duct is to be rasped. The operative procedures advocated are: (1) In acute exacerbation of chronic mastoiditis, modified radical mastoidectomy is used. (2) In acute frontal sinusitis, if intranasal work fails, a sinuotomy of the inferior wall is accomplished, and if osteomyelitic foci of the sinus is to be removed, the anterior and the posterior walls must be taken away to stop the advance of the process. The author recorded a new symptom found in involvement of the temporal bone by osteomyelitis, namely, neuralgia of the upper molar teeth. If the diagnosis of osteomyelitis is made, a very radical procedure with wide resection of the bone should be done.

TOBEY.

Progressive Facial Palsy Produced by Intratemporal Epidermoids.

Jefferson, Geoffrey, (Manchester), and Smalley, A. Ashton (Manchester), J. Laryng. and Otol., 53:417-443 (July), 1938.

Six cases are presented in which there was progressive paralysis of the facial nerve and deafness. In each case an epidermoid (cholesteatoma) was found in the temporal bone, and in no case was any infection found. Three of the patients had never had otitis media. All six cases are considered by the authors to be of embryonal origin.

In all six cases, the facial paralysis was the chief complaint, and in five cases it came on very slowly. All six were markedly deaf and the membrana tympani showed no evidence of existing infection. All six cases showed abnormal caloric responses. In each case, erosion of the temporal bone was clearly demonstrated by x-ray films in the Towne position.

The facial paralysis was relieved in but one case, the only one in which the epidermoid arose from a point external to the fallopian canal.

DEAN, JR.

Experimental Observations on Headache.

Pickering, G. W. (London), Brit. Med. J., 4087:907 (May 6),1939.

The author summarizes and analyses his work and the work of others in the experimental production and relief of headache. Headache produced by histamine, lumbar puncture headache, pyrexial headache, migraine, raised intracranial pressure and traumatic headache were studied and the mechanism of pain discussed.

It is of interest that subarachnoid injection of normal saline solution relieved or abolished the headache of histamine, fever and lumbar puncture. The headache of increased intracranial pressure was always brought on or made worse by withdrawal of cerebrospinal fluid.

Those interested in the mechanism of headache can find in this article many interesting observations and hypotheses too numerous to describe here.

DEAN, JR.

Modern Principles of Treatment in Bronchiectasis. Based Upon 199 Cases Treated by Lobectomy or Total Pneumonectomy.

Edwards, A. Tudor, M.D. (London), Brit. Med. J. 4085:809 (Apr. 22), 1939.

The unsatisfactory results of medical, surgical and bronchoscopic treatment in bronchiectasis are discussed in brief, followed by a more lengthy discussion of the treatment by lobectomy and pneumonectomy.

Reviewing the statistics of 166 lobectomies and 33 pneumonectomies, the author finds the operative mortality zero in children between the ages of four and sixteen years. The mortality increases

as the age group increases, so that early diagnosis helps provide a complete cure at a minimum risk.

Complete bronchography assures removal of all bronchiectatic areas. The lingula of the left upper lobe may be the site of diseased tissue and be left behind in the absence of careful preoperative study.

DEAN, JR.

Abscess of the Brain: Relation of the Histologic to the Clinical Features.

Alpers, Bernard J. (Philadelphia), Arch. Otolaryng., 29:199 (Feb.), 1939.

This article reports a rather exhaustive study of 27 cases of brain abscess, principally from the standpoint of bacteriology and histology, in order to answer the following questions concerning the encapsulation of brain abscesses: (1) How does encapsulation of the abscess take place? (2) When can it be said to have occurred? (3) What relationship does it have to the causative organism? (4) What should its relation be to the operation? The above questions are, for the most part, very satisfactorily answered in the article.

The time, the type of organism and the resistance of the host are the most important factors in the development of the capsule and the optimum time for capsule formation is three to four weeks. Their histologic structure is described, especially with reference to the formation of the capsule of the abscess.

TOBEY.

Notices

Call for the Convention for the Revision of the Pharmacopoeia of the United States of America to Be Held at Washington, D. C., Beginning May 14, 1940

May 1, 1939.

In compliance with the provisions of the Constitution and By-Laws of the United States Pharmacopœial Convention, I hereby invite the several bodies entitled under the Constitution to representation therein to appoint three delegates and three alternates to the Convention for the Revision of the Pharmacopœia of the United States of America, which is to meet in Washington, D. C., on May 14, 1940.

Walter A. Bastedo, M.D.

President of the United States Pharmacopœial Convention.

Biological Photographic Association

The ninth annual Convention of the Biological Photographic Association will be held September 14 to 16, at the Mellon Institute for Industrial Research, Pittsburgh, Pa. The program will be of interest to scientific photographers, scientists who use photography as an aid in their work, teachers in the biological fields, technical experts and serious amateurs. It will include discussions of motion picture and still photography, photomicrography, color and monochrome films, processing, etc., all in the field of scientific illustrating. Up-to-date equipment will be shown in the technical exhibit; and the Print Salon will display the work of many of the leading biological photographers here and abroad.

Further information about the Association and the Convention may be obtained by writing the Secretary of the Biological Photographic Association, University Office, Magee Hospital, Pittsburgh, Pennsylvania.